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THE
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TUMORS OF THE PAROTID GLAND.*†‡

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This analysis of 227 patients with primary tumors of the parotid gland is presented in an effort to add a note of optimism to the prognosis of these lesions. It is indeed difficult to explain the great differences of opinion which exist regarding treatment of these tumors, the marked variation in our ability to eradicate them completely and permanently, and the high incidence of surgical complications after extirpation of a tumor so readily accessible.[1-5]

The pessimistic attitude which has developed has much to support it. A lack of exact knowledge of the anatomy of the facial nerve and its important branches has made the possibility of surgically produced facial palsy a formidable hazard when removal of a parotid tumor is undertaken. Once removed, these tumors are said to recur and with each recur-

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rence an increased rate of growth, increased cellularity and possibly a change from benignancy to malignancy may result.[6,14] As a corollary to this it has been indicated that benefit from excision of such malignant lesions is slight;[6] furthermore, much confusion has arisen from the great variety of terms employed by the pathologist in describing these tumors. His difficulty in distinguishing histologically the benign from the malignant lesions has contributed no little part to the surgeon's difficulty in planning suitable treatment.

The ultimate in pessimistic conservatism has been reached when, on the basis of these reasons, physicians have stated that it is wiser not to operate upon these patients as long as they are willing to wait; that the only unequivocal indications for operation are pain and limitation of mandibular movement; and that if the tumor is carcinoma, "its excision usually fails to afford much benefit."[3,6]

We are of the opinion that the presence of a new growth in the parotid gland is an indication for its removal. In the more than 14 years between July, 1934, and January, 1949, 227 patients with primary tumors of the parotid gland have been admitted to the University of Michigan Hospital. One hundred nineteen of these were treated in the department of otolaryngology and 108 in the department of surgery. Nine of these individuals were seen in consultation only, after operation elsewhere. Some type of operation upon the involved parotid gland was thus carried out on 218 patients in this period. Pathologic material was available on all patients and all have been followed to the present time.

Little value would be derived here from an elaborate discussion of the histogenesis of tumors of the parotid gland. It seems to us that the significance of such studies is often exaggerated. Nor does it seem profitable to enter into an extended defense or discussion of the particular histologic classification used in this hospital.

The majority of patients (113) in this study had tumors of the mixed salivary gland type, which are so classified without further description of the predominant cellular elements

present in each tumor. The second largest group of patients (54) are those whose tumors contained the typical mixed tumor elements, but in which the cellular changes, degree of capsular penetration or extent of infiltration of adjacent parotid gland indicated active infiltrative growth to the extent that they have been classified as carcinomas (or sarcomas) arising in a mixed salivary tumor. The third largest group of patients (41) are those with tumors composed entirely of malignant cells in which any suggestion of origin from the benign mixed tumor is absent. Among the last two histologic groups appear three tumors with the typical pseudoadenomatous basal cell configuration;[7] in two of these, origin in a mixed tumor is apparent. The pathologic material from 16 patients, also included in the malignant tumor groups, was classified as epidermoid or squamous cell in type. In two instances the term mucoepidermoid was used to define the pathologic appearance.[8] One of these latter two patients had distant metastases at the time of his death.

Ten patients in this series had tumors designated as papillary cystadenoma lymphomatosum.[9] This unusual tumor, like the mixed tumor, has a debatable histogenesis. Microscopically, it is composed of tall papillary epithelial projections embedded in lymphoid stroma which may contain germinal centers. It is a wholly benign lesion, more common in men than in women, and its histologic characteristics are unmistakable.

The sarcomas of the parotid gland are rare tumors with no distinguishing clinical characteristics. There exists some doubt[10] whether these are true connective tissue neoplasms or are highly undifferentiated tumors of epithelial origin.

Suspicion exists in the minds of many surgeons that repeated excision of a benign parotid tumor may lead ultimately to malignancy. Such a thesis is difficult to prove, since obvious and unequivocal evidence of this change is seldom seen. We have been able to find only three patients in whom definite change from a benign to a malignant lesion can be demonstrated. In one patient a benign mixed salivary tumor of the

parotid gland was removed in 1928, recurred in 1933 and, when removed again in 1938, was found to be a poorly differentiated medullary carcinoma. A similar instance occurred in another patient 12 years after the appearance of the original tumor and nine years after the first removal. At the second operation a carcinoma, arising in a mixed salivary gland tumor, was found. The third patient's mixed tumor was removed in 1927, recurred in 1939 and was removed here in 1943. At this time carcinomatous change was present within the tumor. A second recurrence, present since December, 1948, has now caused this patient to be readmitted. Such instances must be interpreted guardedly since serial sections of the original tumor might well display evidences of malignancy not apparent in the histologic sections examined. Three additional patients with malignant tumors are known who are reported to have had benign lesions originally, but factual evidence of the benignancy of the early lesion has been unobtainable. Many examples exist, here and elsewhere, of repeated excisions of a benign lesion in which no evidence of malignancy at a later operation was discovered. The evidence that change from a benign to a malignant tumor does occur seems to rest, then, in the tumor known to be present, 10, 15 or 20 years, in which histopathologic evidences of malignancy are seen; however, it seems unlikely that, when malignant, these tumors would remain localized as long as 10 to more than 20 years. Inspection of Table III suggests that when only the known duration of the tumor is considered, there is a rather uniform incidence of all carcinomas in this series up to the four to six-year periods. From our knowledge of these tumors such duration is not unlikely and it may be assumed that malignant elements may be present within the tumors during the entire period. The incidence then falls off as shown in this table until the 10 to 20-year periods are reached. This suggests that a benign tumor had existed 10, 15 or 20 years, that change in its growth or appearance prompted medical consultation and that this change resulted from malignant degeneration in the mixed tumor apparent to the pathologist after removal of the tumor. Similar conclusions might be drawn from the primary carcinoma group

where evidence of a pre-existing benign lesion might be obscured by rapid growth of the malignant cells. The data presented here suggests, therefore, that time, rather than the trauma of repeated operation, may be the greater factor in the appearance of malignancy in a benign mixed tumor.

The clinical manifestations of parotid gland tumors usually make their diagnoses obvious. This is particularly true when the tumor lies in the superficial portion of the gland. Those tumors arising in the gland behind the ramus of the mandible and deep to the facial nerve are less readily defined and infiltration into adjacent gland or fixation to neighboring structures is demonstrated with difficulty. Tumors in the latter area may fill the lateral pharyngeal space and are commonly much larger than anticipated.

The symptoms presented by the patients in this series are varied but were always concerned with the presence of a tumor (see Table I). The occurrence of pain or tenderness

SYMPTOMS

	MIXED TUMORS	CARCINOMA IN MIXED TUMORS	CARCINOMA	SARCOMA IN MIXED TUMORS	SARCOMA	PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM
TUMOR	76	30	17	6	0	6
TUMOR PAIN TENDERNESS	37	16	13	1	1	3
TUMOR BLOOD OR PUS IN SALIVA	0	0	1	0	0	1
TUMOR PAIN FACIAL PALSY	0	2	6	0	1	0
TUMOR ULCERATION	0	4	1	0	0	0
TUMOR FACIAL PALSY SYMPTOMS OF METASTASES	0	2	3	0	0	0

Table I.

over the tumor or radiating over the side of the face was of little value in differentiating the malignant lesions. On the other hand, the appearance of facial palsy not produced by prior surgical trauma always indicated invasion of the nerve

by carcinoma or sarcoma. There was no instance in which a benign tumor produced facial paralysis. The facial nerve withstands stretching to an enormous degree if this is accomplished gradually by a slowly expanding tumor. The presenting symptoms in three patients were related to the presence of distant metastases in bone, lung or mediastinum.

A benign, slowly growing tumor which exhibits suddenly an increased, unexplained growth rate usually is considered to have undergone malignant change. That this is not always true is apparent in the significant proportion of patients with mixed tumors who indicated their awareness of recent rapid growth. The incidence of this change in growth rate is much greater in patients with malignant lesions, but its presence

GROWTH RATE

	RECENTLY INCREASED GROWTH	SLOW GROWTH
MIXED TUMORS	17	96
CARCINOMA IN MIXED TUMORS	14	40
CARCINOMA	13	28
SARCOMA IN MIXED TUMORS	1	6
SARCOMA	1	1
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM	0	10

Table II.

alone did not invariably indicate malignant change (see Table II). The reliability of the patient's observation on rate of

growth is frequently faulty and weighs further against the usefulness of this finding. A change in growth rate with infiltration into adjacent gland or fixation of the more rapidly growing tumor to skin or underlying structures is of more important significance; however, one-third of the patients in this series had had from one to four operations before their admission to this hospital, while another less large group had received some form of irradiation therapy over their tumor, even when benign, thus diminishing considerably the value of any observation on the degree of fixation.

The differentiation between benign and malignant tumors cannot be made on the basis of the duration of symptoms (see Table III). Likewise, little additional help is obtained from

DURATION OF SYMPTOMS
(ONSET TO FIRST ADMISSION)

	0–1 MO.	1–3 MO.	3–6 MO.	6–12 MO.	1–2 YRS.	2–3 YRS.	3–4 YRS.	4–5 YRS.	5–6 YRS.	6–7 YRS.	7–8 YRS.	8–9 YRS.	9–10 YRS.	10–15 YRS.	15–20 YRS.	OVER 20 YRS.	UNKNOWN
MIXED TUMORS	1	2	4	8	14	10	9	10	10	1	4	1	5	8	10	7	9
CARCINOMA IN MIXED TUMORS		2	1	5	5	4	3	3	3	4	1	1	2	9	3	6	2
CARCINOMA		2	7	7	4	2	1			3		2	2	3	2	6	
SARCOMA IN MIXED TUMORS		1				3		1						2			
SARCOMA							1	1									
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM			1	2	1	1	2	1	2								

Table III.

the size of these tumors (see Table IV) since many of the largest tumors were malignant and had been present for many years.

While the correct diagnosis of parotid gland tumors usually is not difficult, the physician must on occasion differentiate chronic inflammatory processes from tumor. In practically

all instances chronic suppurative processes involve the entire gland. In all patients with parotid swelling, the orifice of Stenson's duct must be examined. Gentle massage over the

SIZE OF TUMOR ON ADMISSION

	1 CM.	2 CM.	3 CM.	4 CM.	5 CM.	6 CM.	7 CM.	8 CM.	9 CM.	10 CM.	OVER 10 CM.	NOT RECORDED	REMOVED ELSEWHERE
MIXED TUMORS	9	28	37	8	11	11	2	1	0	1	0	5	0
CARCINOMA IN MIXED TUMORS	2	8	13	3	9	4	1	2	0	2	2	6	2
CARCINOMA	3	4	6	4	6	3	1	2	1	1	0	9	1
SARCOMA IN MIXED TUMORS	0	3	1	0	0	2	0	1	0	0	0	0	0
SARCOMA	0	0	0	0	1	0	0	0	0	0	0	1	0
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM	0	1	3	2	3	1	0	0	0	0	0	0	0

Table IV.

parotid gland and pressure along the course of the duct will result in a flow of fluid from the duct orifice. In the presence of a chronic inflammatory process a thick mucinous fluid or pus will be obtained. In rare instances (see Table I) pus or blood will be expressed from the duct when a tumor is present.

If the patient's history indicates considerable fluctuation in the size of the tumor and if swelling increases at meal time, an obstruction in the duct, probably a calculus, must be considered. In such instances probing of the duct and radiographic studies may facilitate the diagnosis.

Further differentiation must be made between hypertrophy of the masseter muscle and parotid tumors. Within the past two to three years we have become increasingly aware of hypertrophy of this muscle as a clinical entity and a cause of swelling in the parotid region. Four patients have been observed with such a condition simulating a parotid tumor. In each instance the swelling has occurred in a young female

and has been unilateral. Clinical differentiation may be confirmed by the increased prominence of the mass with contraction of the masseter muscle. Two of these patients have had confirmation of this diagnosis by surgical exploration. Further studies on their etiology, character and pathology are in progress.

The parotid gland may be the seat of a lymphoblastoma, primary here or associated with generalized manifestations of the disease. In such instances bilateral parotid tumors may be encountered.

Since the evidence of malignancy in a parotid tumor cannot be determined accurately by the size of the tumor, its rate or duration of growth, or by the age (see Table V) or the sex

AGE

DECADES	2ND	3RD	4TH	5TH	6TH	7TH	8TH	9TH
MIXED TUMORS	8	17	28	21	18	15	6	
CARCINOMA IN MIXED TUMOR	2	6	15	9	11	5	3	3
CARCINOMA	1	4	6	7	5	11	5	2
SARCOMA IN MIXED TUMOR		4	2			1		
SARCOMA			1		1			
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM					3	5	2	

Table V.

(see Table VI) of the patient, the treatment undertaken must be based on the premise that a malignant tumor exists. If cure is to be attempted by surgical resection of any known malignant tumor it must be carried out before distant metastases have occurred. The decision in favor of any form of treatment then depends upon the estimated end-result of that treatment. This estimate is based primarily upon long term survival of the patient without recurrence of the tumor. In

SEX

	MALE	FEMALE
MIXED TUMORS	46	67
CARCINOMA IN MIXED TUMORS	25	29
CARCINOMA	22	19
SARCOMA IN MIXED TUMORS	4	3
SARCOMA	1	1
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM	7	3

Table VI.

SURVIVAL*

		0-2 YRS.	2-5 YRS.	5-10 YRS.	10 YRS.+	TOTAL
MIXED TUMORS	ALIVE	13	41	38	14	106
	DEAD	0 / 2	0 / 1	0 / 1	0 / 0	4
CARCINOMA IN MIXED TUMORS	ALIVE	8	12	11	8	39
	DEAD	4 / 2	1 / 0	0 / 1	1 / 2	11
CARCINOMA	ALIVE	9	3	2	3	17
	DEAD	6 / 1	1 / 0	0 / 1	0 / 0	9
SARCOMA IN MIXED TUMORS	ALIVE	0	0	2	3	5
	DEAD	1 / 0	0 / 0	0 / 0	0 / 0	1
SARCOMA	ALIVE	0	0	0	1	1
	DEAD	0 / 0	0 / 0	1 / 0	0 / 0	1
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM	ALIVE	1	6	1	0	8
	DEAD	0 / 0	0 / 1	0 / 0	0 / 1	2

*AFTER LAST OPERATION AT UNIVERSITY HOSPITAL. DEAD OF TUMOR / DEAD UNRELATED CAUSE

Table VII.

Table VII is shown the survival of the 204 patients included in this study who had their tumors excised. Nine patients were not operated upon in this hospital. Fourteen patients had only a simple biopsy of their tumor mass. There was one operative death from injury to the common carotid artery, in a patient with a far advanced carcinoma arising in a mixed tumor, an immediately operative mortality rate of 0.49 per cent. Six patients of the carcinoma in mixed tumor group and seven patients in the primary carcinoma group have died from recurrence of their neoplasms. Thus 12 per cent of those patients with carcinomas appearing in a mixed tumor and 27 per cent of those with ductal carcinomas are dead of their disease. In this group of patients the majority of deaths from tumor recurrence occurred within the first two years; survival beyond the five-year period gave an excellent expectancy for a permanent cure. The one patient, who died 10 years, nine months after removal of a medullary carcinoma, did so from a similar tumor in the bronchus. Because of the low degree of differentiation of the bronchial neoplasm, its origin, whether from the bronchus or previously removed parotid tumor, could not be determined.

The patients with sarcoma of the parotid are few, and little significance can be attached to their death rates in our series.

From the foregoing table it is apparent that long survival is not unlikely after surgical removal of a malignant parotid tumor. As in patients with neoplasms elsewhere, this favorable outcome is dependent upon the size and extent of the tumor when seen and the thoroughness with which its removal can be accomplished. It does not seem, therefore, that continued observation until such a tumor reaches tremendous proportions may have any commendable virtue.[3]

Recurrence of tumors (including all groups) after resection in this hospital has been seen in 27 patients, an overall incidence of 13.3 per cent. Nine patients have returned for reoperation. Five of these have had one subsequent operation after the initial one, two have had two later operations, and in two patients three subsequent resections have been done.

While recurrence may result from the presence of a satellite tumor which has remained unrecognized after removal of the larger tumor, and while some of the mixed tumors may have multicentric origins, most recurrences have resulted from inadequate removal. A tumor 1 to 2 cm. in diameter is frequently enucleated under local anesthesia and fear of damage to a branch of the facial nerve readily leads to incomplete intracapsular removal. There is little doubt that this is the reason that a higher recurrence rate is noted, particularly in the benign mixed tumors, when small lesions are removed surgically. Ideally, entire visualization of the adjacent branches of the nerve should be obtained and resection of the intact tumor with a wide margin of adjacent parotid gland performed. By such a method only can recurrence after operation be attributed to the presence of a tumor with multiple centers of origin.

Squamous cell carcinomas arising in the parotid gland are considered highly malignant.[10] Sixteen patients in this present survey had lesions of this character. Five of these were treated by Roentgen therapy alone. Two of them might well have been considered suitable for surgical resection at the present writing. These five patients so treated survived from seven to 15 months, with an average survival of 10 months. In 11 patients resection of the tumor and parotid gland was undertaken; three of the patients have been operated upon recently and must await longer evaluation. Five of these 11 patients are now dead, three of them dying with recurrence of their tumors between one and two years afrer excision, and two dying four and one-half and nine years after operation of unrelated causes and without clinical evidence of recurrence. At the time of death three of the patients with squamous cell lesions had distant metastases in spine, lung or mediastinum. The three remaining patients are alive, without clinical recurrence of their tumors after adequate surgical excision, five and one-half to 14 years. Thus five patients, either alive or dead without clinical reappearance of their tumors, survived four and one-half to 14 years after resection.

The incidence of less well differentiated lesions in the squamous cell carcinomas is greater than in the other histologically malignant tumors. Thus nine of the 16 patients with squamous cell carcinomas had Grade III or ungraded medullary lesions.

In the group of 113 patients with benign mixed tumors, there have been 12 patient recurrences (10.9 per cent). Ten patients are now living who have recurrences of their mixed tumors.

In the 10 patients with papilliferous cystadenoma lymphomatosum, no secondary operations have been done and no recurrences are present.

In the entire group of 84 patients with malignant tumors, recurrence after resection has occurred in 15, or 17.8 per cent. Two patients living from five to 15 years after resection of a carcinoma arising in a mixed tumor have recurrences.

The figures presented in the survival table (see Table VII), however, do not give an entirely accurate estimate of prolonged survival in the patients with malignant disease, since approximately one-third of these patients have been operated upon within five years. In this five-year period the greatest number of recurrences and deaths occur and until these individuals have survived without recurrence more than five years, their recurrence rate cannot be estimated with assurance.

The customary evaluation of therapy on the basis of five-year cure is difficult in a series of this type. Tabulation of such cures in the case of benign mixed tumors carries no significance since recurrences have been noted 20, 30 or more years after excision; however, in the case of malignant tumors, a tabulation of five-year survivals without recurrence seems justifiable (see Table VIII). In the 25 patients with carcinoma in mixed tumors, operated upon more than five years ago, 18, or 72 per cent, are alive or lived five years or more without recurrence of their tumors. Of the 11 patients with primary carcinoma who were operated upon more than five years ago, six survived more than five years without

recurrence of tumor. Five of these patients are alive at this time. Five of the six patients with sarcoma arising in a mixed tumor are alive without tumor recurrence more than

FIVE YEAR SURVIVAL AFTER EXCISION

	CARCINOMA IN MIXED TUMORS	CARCINOMA	SARCOMA IN MIXED TUMORS	SARCOMA	TOTAL
ALIVE WITHOUT RECURRENCE OF TUMOR	16 (64%)	5 (45%)	5 (83%)	1 (50%)	27 (61.3%)
DEAD WITHOUT RECURRENCE OF TUMOR	2 (8%)	1	0	0	3 (6.8%)
ALIVE WITH RECURRENCE OF TUMOR	2	0	0	0	2 (4.5%)
DEAD FROM RECURRENCE OF TUMOR	5	5	1	1	12 (27.2%)
TOTAL PATIENTS	25	11	6	2	

Table VIII.

five years after operation. Only one of the two primary sarcoma patients operated upon more than five years ago is alive without recurrence.

Thus 68.1 per cent of the patients operated upon more than five years ago have survived from five to 10 years without recurrence of tumor. Twenty-seven, or 61.3 per cent, of these are alive without recurrence at this time.

The results described thus far are based on the premise that tumors of the parotid gland are best treated by surgical removal. The plan of surgical attack has been relatively uniform. The variations in method are dictated by the size, character and location of the tumor.

In the surgical removal of a parotid tumor, adequate exposure of the field is of paramount importance. Small, incorrectly placed incisions endanger the facial nerve and make

complete excision of the tumor difficult or impossible. The incision usually used begins just below the level of the zygomatic process and immediately in front of the tragus. It is carried below the ear posterior to the angle of the mandible,

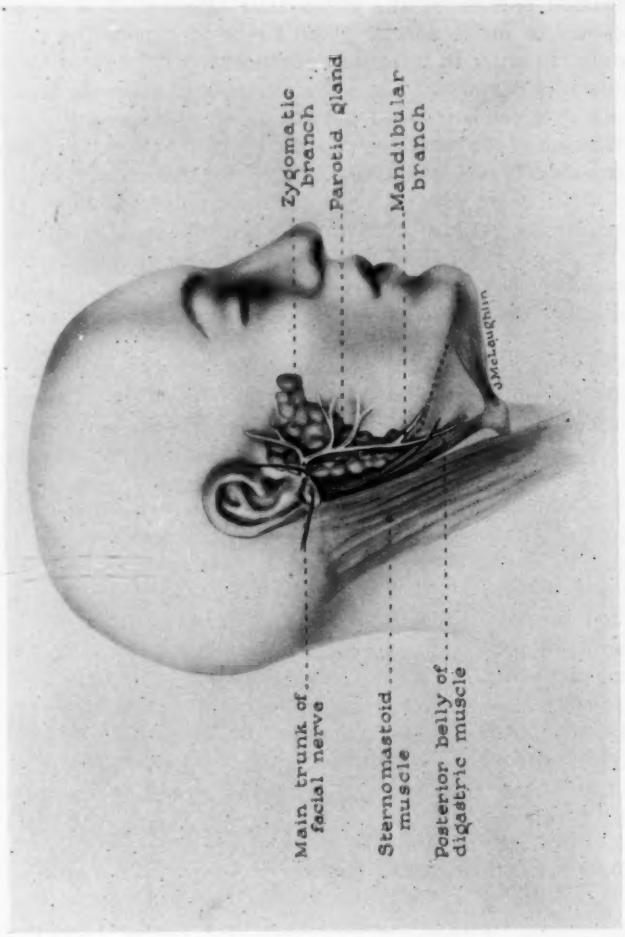

Fig. 1.

and beneath the lobule for 1 to 2 cm., then forward along the posterior belly of the digastric muscle. The skin and subcutaneous tissues of the flap thus formed are reflected forward, exposing the surface of the parotid gland widely. Although the tumor may present itself on the surface of the superficial portion of the gland thus exposed, it is usually necessary to incise normal gland tissue to expose the tumor capsule. In order to safeguard any branch of the facial nerve in this area it is wise to pass the electrode of a simple faradic excitor over the surface of the gland before incising it. If no contraction of the facial muscles occurs with two to three volts of faradic current the operator may be reasonably certain that incision into gland tissue at this point is safe. This maneuver is of utmost importance when the tumor is attacked in the above fashion, since tumors arising in the deep or pterygoid lobe of the gland may produce atrophy of the superficial gland tissue and push the facial nerve far lateralward.

When the capsule of the tumor has been exposed, both the tumor and the capsule must be removed. Intracapsular evacuation of a surgically exposed tumor, followed by curettage of the remaining tumor elements has no place today in the surgical treatment of parotid tumors.[2] Portions of the capsule left *in situ* are often the source of recurrence since small islands of tumor tissue or epithelial ducts may be demonstrated in microscopic sections of the connective tissue capsule. Considerable effort should be made to prevent rupture of the tumor. Occasionally recurrent mixed tumor nodules are found in the subcutaneous scar, indicating that seeding of the tumor at the time of the original excision had occurred. The ease with which mixed tumors of the parotid gland are grown in tissue cultures seems to favor this hypothesis.[11] When soiling of the wound by spilled tumor contents occurs, vigorous mechanical cleansing by prolonged irrigation should be done.

Tumors in the superficial portion of the gland may lie on the facial nerve or its branches. As the tumor is elevated from its bed, "tenting" of the nerve may result. Care must

be taken in freeing the mesial portion of the tumor lest a segment of the nerve be excised.

Tumors lying in the deeper portions of the parotid gland, mesial to or beneath the facial nerve, demand exacting technique in their removal. In such instances the operation

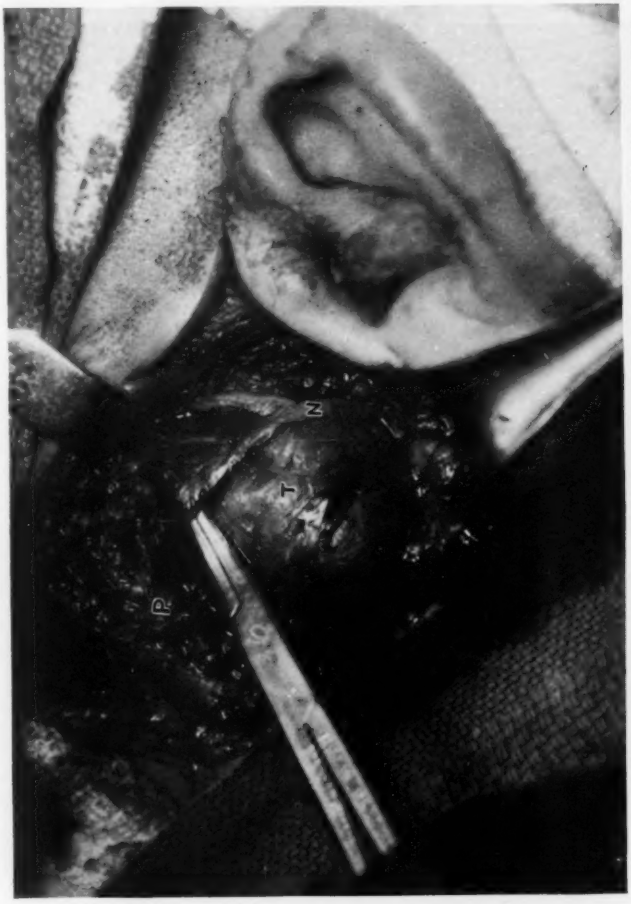

Fig. 2. Facial nerve (N) pes anserinus and the temporofacial and cervicofacial divisions greatly stretched over a deeply lying tumor (T). Residual parotid gland (P) lies anterior and superficial to the more distal nerve branchings.

becomes an anatomical dissection of the facial nerve and its branches with incidental removal of the tumor or perhaps the entire parotid gland. If the facial nerve has been stretched gradually by the expanding tumor, it will withstand considerably more surgical trauma than normally.

If the tumor is very large, its removal may be facilitated

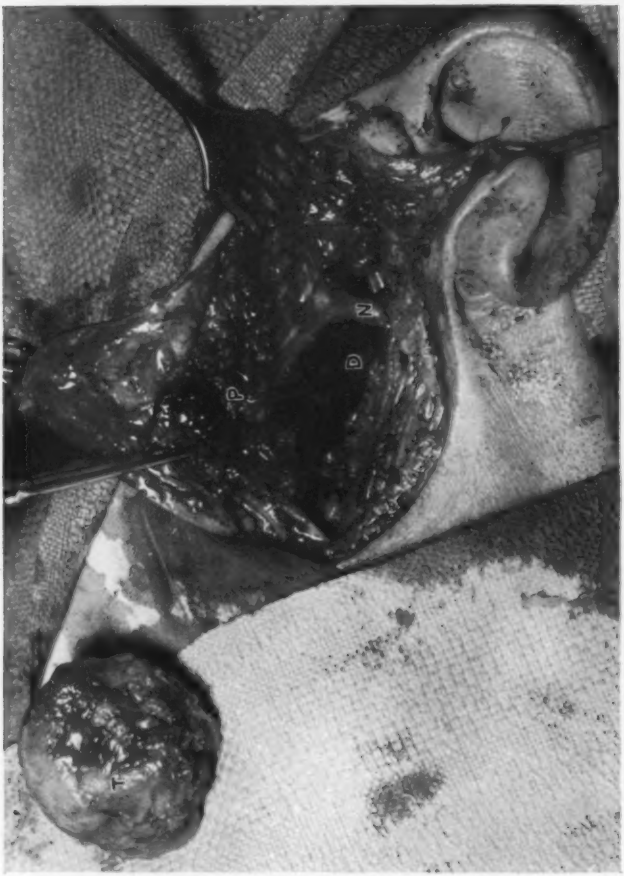

Fig. 3. Tumor (T) has been removed, leaving large defect (D) below the facial nerve (N) which has assumed its normal position.

by opening the capsule and removing a portion of its contents. This will permit removal of the entire capsule even if it has invaded the lateral pharyngeal space.

Since tumors of large size often distort the normal course

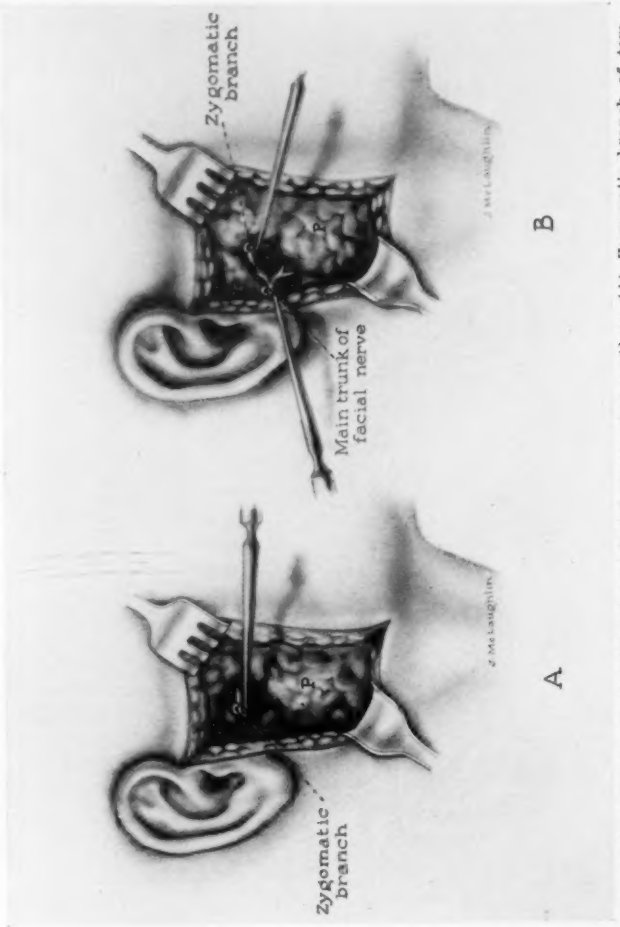

Fig. 4. Technique I for identification of facial nerve at operation; (A) Zygomatic branch of temporofacial division of the facial nerve exposed through incision in parotid gland (P) parallel to lower border of zygomatic arch. (B) Proximal exposure of zygomatic branch, pes anserinus and main facial trunk.

of the facial nerve, the surgical approach to such a tumor may be facilitated by preliminary exposure of the nerve. The initial exposure of the facial nerve may be accomplished in various ways:

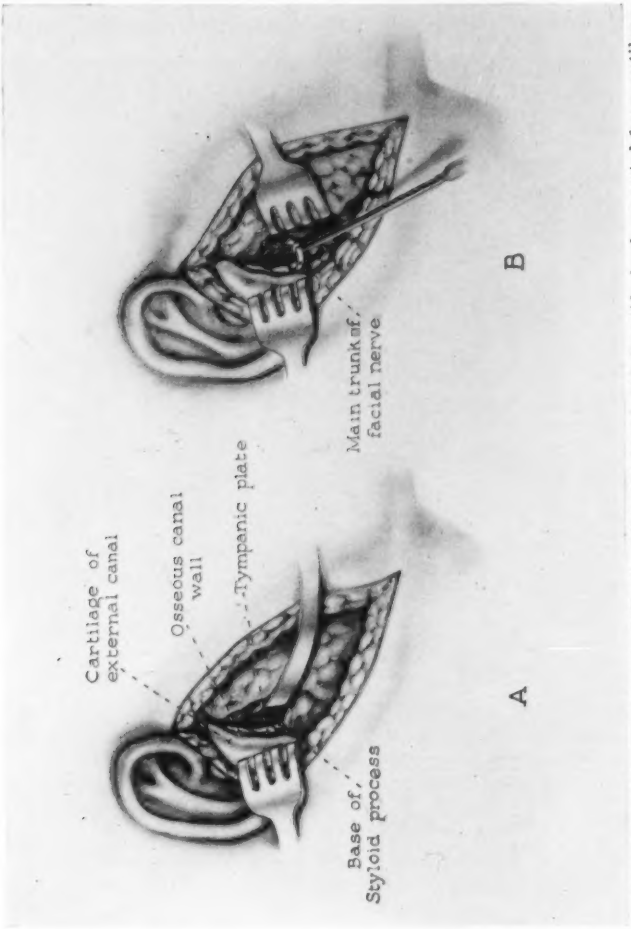

Fig. 5. Technique IV for identification of facial nerve. (A) Parotid gland separated from cartilage of external ear and osseous canal wall. Tympanic plate and base of styloid process exposed. (B) Main trunk of facial nerve identified as it crosses base of styloid process.

1. After reflecting the skin flap, the zygomatic branch of the nerve which lies inferior and parallel to the zygomatic arch may be located beneath the surface of the gland. This branch may then be traced posteriorly to expose the pes and

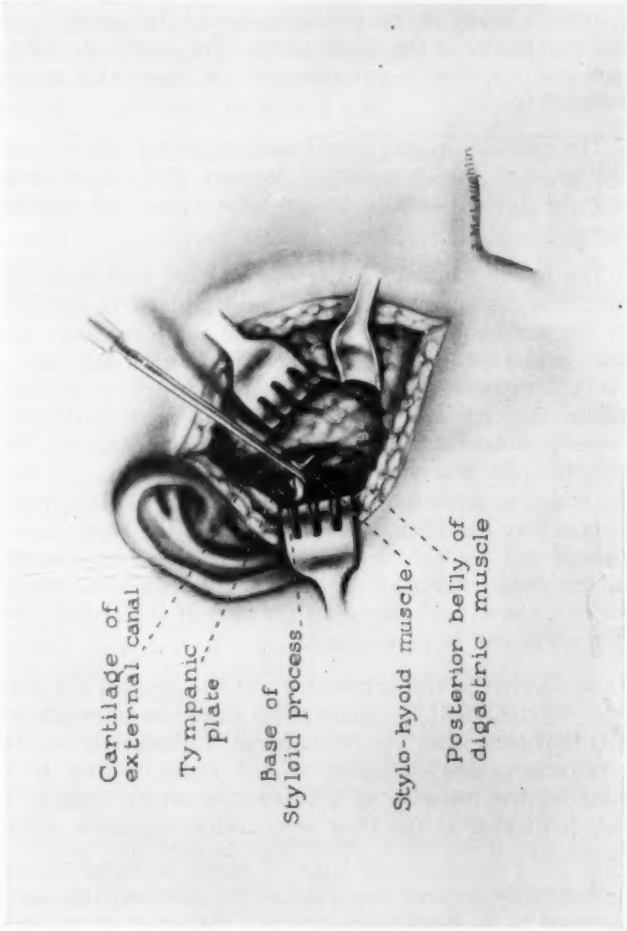

Fig. 6.

the main facial trunk. This method of identification is best used when the tumor is large and encroaches upon the stylomastoid foramen, making identification of the nerve as it leaves the foramen hazardous.

2. The mandibular branch of the nerve may be identified as it extends below the horizontal ramus of the mandible and traced proximally to the facial trunk. The small size of this branch and its slightly variable position make this method less desirable.

3. The mastoid tip may be removed to permit identification of the nerve at the stylomastoid foramen, from which point it may be traced distally to its bifurcation and terminal branches.

4. The fourth method[13] is the one we have used most commonly: the fascial sheath of the parotid gland is separated from the perichondrium of the external auditory canal. Dissection is carried past the tympanic plate until the base of the styloid process is identified. This dissection is relatively bloodless. The trunk of the facial nerve is next identified as it passes just lateral to the base of the styloid process. Frequently the region between the mastoid process and the angle of the mandible is filled by tumor and isolation of the nerve at this stage may be difficult. In this instance the lower pole of the gland and the enclosed tumor may be elevated readily from the underlying posterior belly of the digastric muscle and as the dissection approaches the base of the styloid process the nerve will be encountered.

When a tumor is unencapsulated and has invaded the gland widely, extirpation of the entire gland should be accomplished. This is best done after the facial nerve is completely exposed and retracted. Any branches of the nerve, known to be invaded by the presence of preoperative muscle palsies, or grossly infiltrated at the time of operative exposure, should be sacrificed.

Inadvertently severed facial branches, or those transected deliberately to facilitate removal of a tumor, must be reap-

proximated accurately at the end of the operation. The prognosis for return of function of the facial nerve is better when the main trunk is severed and resutured than when it is stretched to the point of severe injury in attempting to extirpate tumors of large size.

Far advanced malignant tumors of the parotid gland may produce metastatic foci in the regional cervical nodes. In the absence of far distant spread, radical removal of the deep cervical nodes should be carried out.

The results of surgical treatment, carried out in the manner just described, are measured not only in the number of patients free from recurrence of their tumor and the length of survival of patients with malignant lesions, but also in the number and kind of complications directly related to this method of treatment. In Table IX are listed these sequelae.

POSTOPERATIVE COMPLICATIONS

	TOTAL PATIENTS	NO OPERATION	BIOPSY	TOTAL RESECTION	PERMANENT PALSY		TEMPORARY PALSY	SALIVARY FISTULA	RECURRENCE AFTER LAST OPERATION	PATIENT RECURRENCE
					PARTIAL	COMPLETE				
MIXED TUMORS	113	3	0	116	6	2	15	6	10	12 (10.9 %)
CARCINOMA IN MIXED TUMORS	54	4	2	50	9	6	2	0	6	6 (12.5 %)
CARCINOMA	41	3	12	32	4	3	4	0	7	7 (26.9 %)
SARCOMA IN MIXED TUMORS	7	1	0	6	0	0	0	0	1	1 (16.6 %)
SARCOMA	2	0	0	4	0	0	0	0	1	1 (50.0 %)
PAPILLIFEROUS CYSTADENOMA LYMPHOMATOSUM	10	0	0	10	0	0	2	0	0	0
TOTAL	227	11	14	218	19*	11†	23	6	25	27 (13.3 %)

* 10 SACRIFICED DELIBERATELY
† 7 SACRIFICED DELIBERATELY

Table IX.

The most feared complication of surgical removal of a parotid tumor or gland is facial muscle palsy from damage to the facial nerve. This occurred in 53 patients. In 23 of these, the palsy was temporary and return of function of

the facial muscles usually occurred before the patients were discharged from the hospital. In 30, or 14.7 per cent, of the 204 patients whose tumors were removed, some degree of permanent palsy exists. In 17 patients voluntary sacrifice of all or a part of the nerve was necessary because of the size of the tumor or invasion of the nerve by it. Accidental or involuntary paralysis of facial muscles occurred in 6.3 per cent, or 13 patients. In only one instance did total accidental palsy occur and this undoubtedly resulted from severe stretching of the main facial trunk. The great majority of patients who now have some degree of permanent loss of facial muscle function have had injury to the mandibular nerve. Such paralyses are commonly disregarded by the patient and are noted only on examination by the physician.

Post operative salivary fistulae were noted in six patients, all with mixed tumors. All fistulae were temporary and all but two ceased spontaneously. In two patients 450 r. of Roentgen therapy were administered over the parotid gland before the accumulation of salivary secretion ceased. The removal of small tumors near the anterior edge of the parotid gland may result in transection of its duct. Reimplantation into the mouth is readily accomplished if secretion is not arrested by Roentgen therapy.

The place of Roentgen therapy in the treatment of parotid tumors is still not well defined.[12] The majority of mixed tumors are but slightly radiosensitive; nevertheless, many favorable reports exist on the combined use of surgery and Roentgen therapy in these tumors. The arguments for the use of combined therapy are based on the fact that these tumors are not homogeneous and that even though the myxomatous and cartilage-like elements are insensitive to radiation, the epithelial elements may not be. Radiation, by its effect on these elements, should bring about cessation of growth. Since there is little evidence that complete destruction of a mixed tumor is obtained short of extensive damage to the adjacent normal structures, radiation has been used in none of our patients with mixed tumors, either pre- or postoperatively, except in two patients so treated because of salivary fis-

tulae. We are unable to evaluate the effectiveness of such treatment, therefore, on the incidence of subcutaneous recurrence from tumor cells which may have escaped after rupture during surgical extirpation.

The radiosensitivity of malignant parotid gland tumors is seldom marked, although Ahlbom[12] found that 16 per cent of his patients' tumors showed marked sensitivity. Among his patients the pseudoadenomatous basal cell carcinomas showed good radiosensitivity, while the most resistant tumors were the adenocarcinoma and squamous cell carcinoma types.

	Post-operative Roentgen Therapy	Dead	Alive	No Post-operative Therapy	Dead	Alive
Carcinoma in mixed tumors.....	19	4	15	29	6	23
Carcinoma	18	8	10	8	1	7
Sarcoma in mixed tumors.....	2	1	1	4	0	4
Sarcoma	1	0	1	1	1	0

Forty patients with malignant tumors, upon whom we carried out surgical resection of the tumor, were given Roentgen therapy postoperatively. The distribution of these patients and their present survival is indicated above. This is a small number of patients and the reasons for or against administration of therapy in the postoperative period cannot be correctly evaluated at this date.

In general, the administration of such treatment at the present time depends upon the degree and extent of invasion of adjacent tissues by the tumor and the thoroughness with which the surgeon is able to ablate it. When given at all, Roentgen ray administration is carried to the limits of tolerance of the normal tissues. When complete surgical removal is accomplished, postoperative irradiation is of doubtful value. This concept is particularly applicable in the patients with

malignant change in a mixed tumor since these tumors frequently appear well encapsulated and thus complete removal with a surrounding layer of normal gland is readily accomplished. For this reason the largest number of patients given no postoperative irradiation fall in this category. On the other hand, since complete removal of the widely and irregularly infiltrating carcinomas and sarcomas is less certain, these should receive postoperative X-ray therapy. Without a more accurate and detailed evaluation of the lesions treated by irradiation postoperatively than that possible at this time, we are unable to evaluate properly the usefulness or effectiveness of Roentgen therapy in primary malignant parotid tumors. We are of the opinion, however, that the use of irradiation alone in the treatment of such lesions rarely is curative, and that its use, in conjunction with surgical removal, is of value only where large and vigorous dosage is administered.

SUMMARY AND CONCLUSIONS.

A series of 227 patients with primary tumors of the parotid gland has been recorded.

Statistical study has been made of the 204 patients upon whom surgical excision of the tumors was performed between July, 1934, and January, 1949. Pathologic material was available on all patients and all have been followed to the present time.

In the classification of these tumors six categories were used: One hundred thirteen patients had benign mixed tumors; 10 had a papilliferous cystadenoma lymphomatosum, also a benign lesion; 95 patients had carcinoma, 54 of which appeared to arise in a previously existing benign mixed tumor, while the remaining 41 were considered to be of ductal origin since there was no histopathologic evidence of benign mixed tumor. Nine patients had sarcomas. One hundred twenty of the 123 patients with benign tumors had excision of their lesions. Eighty-four of the 104 patients with malignant

tumors had these tumors excised. Fourteen patients were subjected to biopsy only and nine patients were seen in consultation after previous operations elsewhere.

The duration of the tumor in this series of patients varied from one month to more than 20 years.

A study of the clinical manifestations of these tumors indicates that preoperative facial paralysis and evidence of disseminated metastases are the only reliable signs of malignancy; however, many of the malignant tumors were well encapsulated and showed no evidence of local infiltration or regional spread.

It was not possible in this study to confirm the commonly held opinion that in each successive recurrence of an excised benign mixed tumor, the histologic structure becomes more cellular and the characteristics more malignant. Rather, the indications are that time rather than surgical trauma is the important factor in the development of malignant change in a benign mixed tumor.

Pessimistic attitudes toward the prognosis of patients with parotid tumors are cited and refuted on the basis of the tabulated results of surgical excision of the tumors in this series of patients.

There was one operative fatality in a patient with far advanced carcinoma who died after injury to the carotid artery.

Twenty-seven patients in the entire series have had one or more recurrences after operation; an overall incidence of 13.3 per cent patient recurrences for all tumors. Reoperation has been performed on nine of these patients.

Of the 110 patients who had operative removal of benign mixed tumors, there have been 12 recurrences, or 10.9 per cent. Fifty-seven of these patients are less than five years postoperative. Tabulation of five-year cures in the case of benign mixed tumors bears little significance since recurrences have been noted 20 and 30 years after excision.

In the 10 patients who had excision of a papilliferous cystadenoma lymphomatosum no recurrences have been found.

Of the 50 patients who had excision of the carcinoma arising in a mixed tumor, 25 had the operation more than five years ago. Eighteen of these, or 72 per cent, survived five years or more without recurrence of tumor.

All of the six patients who had operation for removal of sarcoma arising in a mixed tumor were operated upon more than five years ago. Five, or 83 per cent, of these survived five or more years without recurrence of tumor.

Of the 26 patients who had primary carcinomas removed, 11 had their operations more than five years ago. Six, or 54 per cent, of these survived five or more years without recurrence of tumor.

The two patients with primary sarcoma of the gland were operated upon more than five years ago. One of these survived more than five years without recurrence of tumor.

Therefore, of the 44 patients who had operations for the removal of malignant tumors of all types more than five years ago, 30, or 68.1 per cent, survived five or more years without recurrence of tumor.

Twelve per cent of the entire group of patients with carcinoma arising in a mixed tumor and 27 per cent of the patients with primary carconimas are dead of their disease after surgical resection. The primary carcinoma seems to be, therefore, the most malignant type of tumor encountered in this series.

The technique of surgical removal is described, including various methods of identifying and isolating the facial nerve. Every effort should be made to preserve the integrity of the facial nerve when it has not been invaded by neoplasm, as indicated by preoperative paralysis or gross evidence of infiltration at the time of operation.

The use of Roentgen therapy in the treatment of parotid tumors is discussed. This form of therapy should be used in

maximum dosage after removal of poorly encapsulated tumors, the extent of which is not clearly defined, and when total removal is in doubt. Its use alone in the primary treatment of malignant lesions of the parotid gland is rarely curative and is acceptable only when surgical removal is contraindicated.

Fifty-three patients had some degree of facial palsy postoperatively. In 23 instances this palsy was temporary, usually disappearing by the time the patient left the hospital. In 30 patients, or 14.7 per cent, of the 204 patients there was some degree of permanent paralysis due in most instances to deliberate sacrifice of that portion of the nerve involved by the neoplasm. In 13 patients (6.3 per cent) there was accidental or unpremeditated permanent palsy. Only one of this group has complete paralysis. The vast majority of these accidental permanent palsies involve only the lower lip and are of little concern to the patient.

Salivary fistulae occurred postoperatively in six patients. All of these healed promptly without surgical treatment and all occurred in patients who had removal of benign mixed tumors.

The results here tabulated seem to justify the conclusion that all primary tumors of the parotid gland should be removed surgically as soon as they are discovered unless there has been dissemination of metastases to distant structures.

Since the prognosis for survival in patients with parotid tumors depends upon the presence and degree of malignancy and the completeness with which resection of that tumor can be accomplished, and since the differentiation between benign and malignant lesions is not always apparent unless distant metastases have occurred or facial palsy is present, early operation with complete removal of the tumor is imperative.

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TRACHEAL OBSTRUCTION.*†

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The phenomenon of partial obstruction of the trachea is encountered much less frequently than that of laryngeal or bronchial obstruction. In the individual case, however, the implications of this clinical finding are likely to be of some urgency, depending upon the rapidity with which the obstructive dyspnea increases. Obstruction occurring as a result of trauma or the inhalation of certain types of vegetal foreign body may be rapidly fatal unless emergency treatment is available. Cases in which the onset of symptoms is less abrupt require early and accurate diagnosis, so that the occurrence of dangerous degrees of respiratory embarrassment may be averted.

Anatomically, the trachea provides a cross-section area about four times that required for quiet breathing. That this is true, at least in the adult, may be proven by the simple experiment of breathing through a length of tubing, 10 to 12 cm. in length, whose internal diameter, 6 to 7 mm., is approximately half that of the average adult trachea during expiration. As a matter of fact, the resistance to air flow through a normal adult trachea is estimated to be about one-sixteenth that offered by such a length of tubing, in accordance with Poiseuille's Law, which states that the resistance to flow of gases through tubular structures varies inversely with the

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fourth power of the internal diameters, other factors being equal. Air exchange during maximum effort has been estimated as from five to 12 times that of quiet breathing.

According to Jackson, the movements of the trachea may be classified as respiratory, pulsatory, bechic and deglutitory. Physiologically, the inspiratory widening and expiratory narrowing of the trachea is of great importance in explaining the expiratory character of many types of tracheal dyspnea. The expiratory narrowing, easily observed bronchoscopically, and manifested by a forward mounding of the posterior or membranous portion of the tracheal wall, is most pronounced in the intrathoracic portion of the trachea, and appears strikingly in conditions associated with a high positive expiratory intrathoracic pressure, such as bronchial asthma or diffuse pulmonary emphysema.

Symptomatology. In general, partial tracheal obstruction is characterized by expiratory wheezing and dyspnea. The distinctive character of the wheeze heard at the open mouth is such that Jackson[14] many years ago described the "asthmatoid wheeze" as pathognomonic of partial obstruction of the trachea or a large bronchus; the sign has been of particular value in foreign body diagnosis. The wheeze is best heard near the end of a forced expiration; in the lesser degrees of obstruction, wheezing may be observed by the patient only during vigorous physical activity. Dyspnea is evident as the degree of obstruction increases, and the wheeze may become audible during both phases of respiration. Other symptoms associated with partial tracheal obstruction are described below in discussing the various disease entities.

Diagnosis. As has been inferred, the distinctive character of the wheeze, if present, is diagnostic; the nature of the obstruction, however, must be determined. In eliciting the history, the possibility of foreign body should be investigated by specific questioning. If the patient is an infant or young child, the relation between time of birth and onset of symptoms is usually significant. The presence of hoarseness, difficulty in swallowing or "choking attacks" should be noted, and

inquiry should be made as to such associated symptoms as hemoptysis, chronic cough, expectoration, etc. Information regarding previous illnesses and a "systemic review" to elicit symptoms referable to other systems is in order.

The physical examination, in addition to careful observation of the character of any wheeze or dyspnea, and its relation to phase of respiration, should include mirror laryngoscopy; of particular importance in the external examination are the presence of enlarged cervical nodes, localized or diffuse enlargement of the thyroid gland, tracheal deviation or "tracheal tug" and venous distention.

Careful Roentgen examination of the neck and chest (including fluoroscopy and a study of the "swallowing function" with opaque mixture), followed by direct laryngoscopy and tracheoscopy, will then confirm or disprove the presence of tracheal obstruction and in most cases establish the etiology.

"Tracheal Stridor" in Infancy and Childhood. Noisy breath- in infancy and childhood may be a manifestation of obstruction anywhere from the external nares to the smaller bronchi. The "stridor" of *congenital tracheomalacia,* a term implying a deficiency in the support provided by the cartilaginous rings of the trachea, in many instances is of a stertorous character, the expiratory sound resembling that of snoring respirations. A similar sound may be produced by blowing gently through thin-walled Penrose rubber tubing. There is some evidence that increase in the normal expiratory angulation of the infant cervical trachea may be an additional factor in such cases; in several instances, the appearance of suprasternal bulging, synchronous with expiration, has suggested that undue laxity of the supporting tissues at the thoracic inlet may allow increased upward displacement of the upper mediastinal structures with the positive intrathoracic pressure of expiration, thus contributing to the "kinking" effect.

Thymic compression of the trachea, once thought to be of common occurrence, is now regarded as comparatively infrequent. The classic description of the Roentgen findings in this

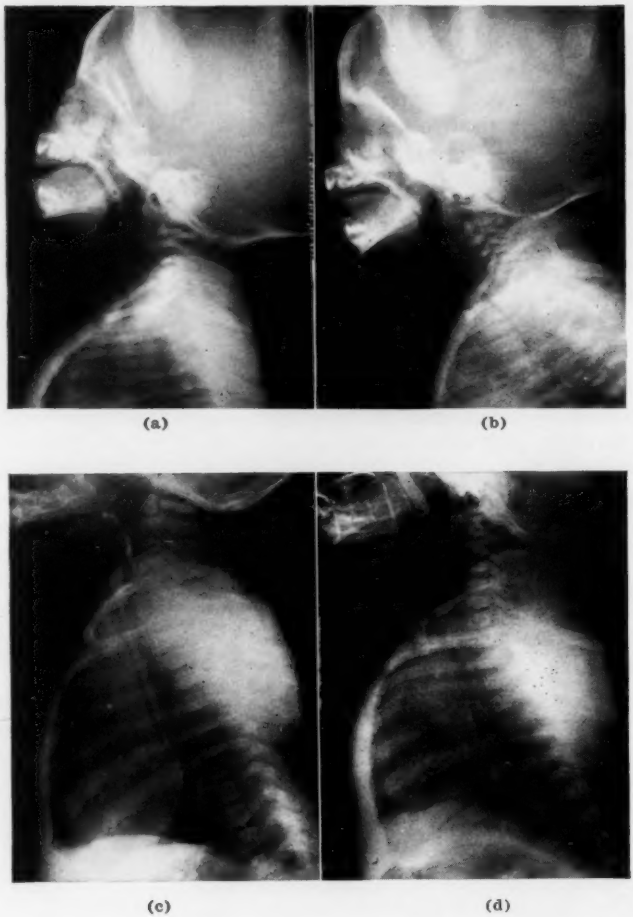
condition is that of Pancoast.[30] The expiratory sound is a true wheeze comparable to that heard in other types of tracheal compression.

In both of the above conditions, the symptoms are likely to date from birth or shortly thereafter, and the noisy breathing is accentuated by activity and by feeding. Unlike congenital laryngeal stridor, however, the respirations during sleep are usually quiet or nearly so, and indrawing of the supra- and infrasternal spaces is less prominent. Roentgen films made during expiration [see Fig. I (b) and (d)] will show, in both conditions, narrowing of the tracheal lumen; however, in tracheomalacia, the inspiration film shows a tracheal lumen which is straight and of normal calibre [see Fig. I (a)]; in true thymic compression, the lumen of the trachea remains narrow even on full inspiration [see Fig. I (c)] and is displaced dorsally.

In tracheomalacia, the tracheoscopic picture is one of tracheal expiratory collapse, with reopening to normal calibre on inspiration. In thymic compression, the narrowing persists in some degree during inspiration, though increased during expiration. If the bronchoscope is introduced to the carinal level, the expiratory wheeze is no longer evident; as it is withdrawn a short distance, the wheeze will reappear. The direct laryngoscopic findings allow a differentiation of these two conditions from laryngomalacia (congenital laryngeal stridor) in which the stridor is nearly always in the inspiratory phase.

The noisy breathing of tracheomalacia is "outgrown" in a few months, or at most two years. In thymic compression, small doses of Roentgen irradiation accomplish regression, and such treatment is indicated once the diagnosis is established or strongly suspected.

A number of recent papers[12,26,36] have called attention to *anomalous vascular ring* as a cause of partial tracheal and esophageal obstruction in childhood. These anomalies, which occur through persistence of embryonal arches other than those involved in the formation of the normally situated



(a) (b)

(c) (d)

Fig. I. (a) and (b) "Tracheomalacia" in an infant of five weeks. Expiratory stridor since birth. (a) Inspiration film, showing tracheal lumen of normal calibre throughout. (b) Expiration film, showing marked narrowing of intrathoracic portion of trachea. (c) and (d) Thymic compression of trachea in an infant of three months. Expiratory wheezing since shortly after birth. (c) The thoracic portion of the trachea is compressed and displaced backward, even in the inspiration film. (d) Narrowing and displacement are increased during expiration.

mature arteries, are, according to Gross[12] of two general types. In one type (right aortic arch, posterior type), the arch, instead of passing from right to left anterior to the trachea, passes from right to left behind the esophagus; constriction of both esophagus and trachea may occur when the pulmonary artery, lying anterior to the trachea, is joined to the distal portion of the arch by a patent ductus arteriosus or ligamentum arteriosum lying to the left of the trachea. The second type is a double aortic arch, the two limbs of the split aorta encircling both trachea and esophagus and reuniting in the left posterior mediastinum. Numerous variations of these types have been described.

The symptoms are wheezing and dyspnea of varying degree (increased during activity and decreased during sleep), and dysphagia or regurgitation, often first noted when an attempt is made to give solid or semisolid foods. The characteristic Roentgen findings (see Fig. II) are clearly summarized by Neuhauser;[26] most suggestive is the finding of a rounded indentation of the posterior wall of the barium-filled esophagus; compression of the trachea may be observed in both projections. A definite diagnosis may be established in nearly all of these cases if Roentgen examination of the esophagus with barium mixture is made a part of the routine study in cases of stridor in infancy and childhood.

Since the tracheal obstruction is at a low level, tracheotomy is not an appropriate palliative procedure for relief of dyspnea, although introduction of a bronchoscope to the carinal level will temporarily provide an adequate airway. Persistent or recurring dyspnea constitutes an indication for thoracotomy, with ligation and division of the anomalous structures responsible for the compression.

The incidence of *tracheal foreign body* is considerably less than that of foreign body in the bronchus. In the author's series of 250 consecutive foreign bodies in the air and food passages,[28] for example, only seven, or 2.8 per cent, were in the trachea, as compared to 46, or 18.4 per cent, in the bronchus. In this group of cases, all of the patients were children, the youngest 11 months and the oldest five years.

Fig. 11. Compression of trachea and esophagus by "vascular ring." Four-year-old girl with history of dyspnea, wheezing and regurgitation, first noted at age of about 10 months. Particularly characteristic is the posterior filling-defect in the barium filled esophagus. The anomaly here is presumably a "right aortic arch, posterior type."

A foreign body small enough to pass through the subglottic lumen would not be expected, initially, to obstruct the tracheal lumen to a dangerous degree, unless of unusual shape or consistency; however, it is generally recognized that foreign bodies capable of increasing in size with absorption of moisture (particularly the bean) may give rise to rapidly increasing dyspnea and possibly asphyxia.

The initial episode of choking, coughing, wheezing and dyspnea may be followed by an asymptomatic interval. More commonly, however, there is a persistent "asthmatoid wheeze." As pointed out by Jackson, foreign bodies freely movable in the trachea may give rise to severe paroxysms of cough; on cough, the "audible slap" and "palpable thud" due to sudden arrest of the foreign body at the subglottic level may be observed.

During cough, impaction in the subglottic region may occur; this event is attended by an abrupt onset of laryngeal dyspnea with inspiratory-expiratory stridor and marked supra- and infrasternal retraction, so that asphyxia appears imminent. In the case of a three-year-old boy recently observed, no foreign body history could be elicited; the symptoms were those of "head and chest cold" of three days' duration. When first seen in the emergency ward, the dyspnea, two-way stridor and retraction suggested acute laryngotracheobronchitis as the most likely diagnosis, but as the bronchoscope was introduced preliminary to tracheotomy, an irregular fragment of peanut was encountered at the subglottic level; this was removed and tracheotomy was not required.

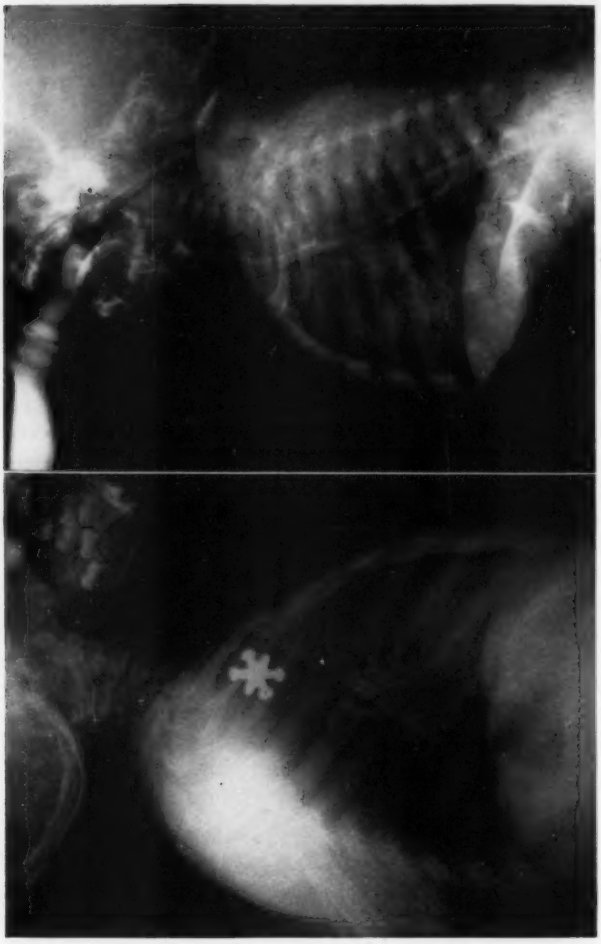
The Roentgen findings in tracheal foreign body may be those of bilateral obstructive emphysema, but in a rather high percentage of cases, even careful fluoroscopic study may fail to show restricted motion of the leaves of the diaphragm or impairment of emptying of the lungs. Such was true in the case of a two and one-half year old boy whose wheeze, present for 18 months before his first visit to the bronchoscopic clinic, had occasional X-ray therapy for a supposedly enlarged thymus, tonsillectomy and allergic study under an erroneous

diagnosis of bronchial asthma. The wheeze disappeared completely with bronchoscopic removal of a wooden bead from the trachea.

Tracheal wheeze and dyspnea may occur as a result of compression of the thin "party wall" between the trachea and esophagus by foreign body lodged in the upper esophagus [see Fig. III (a)]. If opaque, the lateral Roentgenogram will show its true location. If nonopaque, the presence of wheezing may appear to call for bronchoscopy, unless the presence of esophageal foreign body has been demonstrated by careful Roentgen study with barium mixture.

Endogenous foreign body is a common cause of tracheal obstruction in the acute tracheobronchial infections of childhood. Rapidly increasing dyspnea may occur in the presence of diphtheritic exudate or the thick, partially crusted secretions characteristic of acute laryngotracheobronchitis. As emphasized by Jackson[18] and others, the formation of crusts following tracheotomy may usually be prevented by proper humidification of the inspired air and frequent catheter aspiration through the tracheal cannula, with preliminary instillation of a few drops of physiological saline, 1 per cent sodium bicarbonate solution, or a solution of penicillin in saline (5,000 u/ cc.). The persistence, following thorough aspiration, of wheezing, dyspnea or impaired aeration as determined by auscultation, suggests the presence of residual crusted material, and bronchoscopic removal may be required. Similar problems may arise in instances of acute infection following tracheotomy for other conditions and following laryngectomy.

Chronic Granulomata. In tuberculosis, lesions of the trachea are less frequent than those of the larynx and bronchi. More often than not, the tracheal lesion represents proximal extension of one initially involving the bronchi. The stages of the disease are the same as those of tuberculous bronchitis[24] (submucous infiltration, ulceration or ulcerogranuloma, cicatricial stenosis), but tracheal obstruction due to granuloma is uncommon, and cicatricial stenosis is much less



(a) (b)

Fig. III. (a) Compression of trachea by foreign body (toy jackstone) in upper œsophagus of a child of two years. (b) Tracheal compression due to periœsophageal cellulitis. Infant of six months with cough, unexplained wheezing and dyspnœa of three weeks' duration. Œsophagoscopy showed a slender fragment of pearl button in the cervical œsophagus. Resolution following removal of the foreign body; external drainage not required.

common than in the bronchus, presumably because of the infrequency with which a large portion of the circumference of the tracheal wall is involved in the ulcerative stage. Sweany and Behm[35] observed tracheal involvement in only 10.3 per

cent of 377 cases of tuberculous tracheobronchitis. In a study of 22 cases in which perforation of a tuberculous lymph node had occurred, Auerbach[1] found six instances of perforation into the trachea.

The diagnosis of active tuberculous tracheitis is suggested by the tracheoscopic appearance of superficial ulceration, with or without granuloma, and the concomitant finding of Roentgen evidence of pulmonary tuberculosis and positive sputum. Active tuberculous tracheitis in the absence of parenchymal disease is rare. Still more conclusive proof is obtained by removal of a small specimen of tissue for biopsy; the author has seen four cases in which histologic study showed coincident carcinoma in the presence of active pulmonary tuberculosis.

Superficial ulcerations may be treated biweekly by local application of silver nitrate (10 to 20 per cent); granuloma may require forceps removal or electrocoagulation. Rapid healing may follow administration of streptomycin systemically and by inhalation (0.20 gm. in 1 cc. of physiological saline four or five times daily).

Ulcerative or gummatous *luetic lesions* are rare. The finding of atypical ulceration in the presence of positive serologic test, however, should call for biopsy and dark-field examination of material obtained by swab at the time of tracheoscopy.

Tracheobronchial lesions of *scleroma* occur in about 9 per cent of cases, according to the statistics of Belinoff.[2] This disease, endemic in central and southeastern Europe, and recently in Latin American countries, is occasionally encountered among the native-born of the United States. Although generally thought to be of infectious origin, the primary etiologic rôle of the von Frisch bacillus, which can nearly always be cultured from the lesions, has not been fully proven. Grossly, the lesions may be of diffuse or circumscribed character, with indurative thickening of mucous membrane and subjacent tissues, whose consistency is often that of scar tissue

or cartilage. Microscopically, the characteristic feature is the presence of Mikulioz cells, within which the bacilli may be found.

The laryngotracheal symptoms are those of obstruction, which may be partiallly the result of crusted secretions adherent to the dry mucosal surface. Planigraphic study, or films made following instillation of lipiodol (see Fig. IV) may show well the extent and degree of the stenosis. The immediate indication for treatment may be relief of obstructive dyspnea by tracheotomy. Periodic peroral dilatation may prevent occurrence of stenosis in less advanced cases. Jackson advocates that patients residing in a region where the disease is endemic should be encouraged to move elsewhere.

Response to Roentgen or radium irradiation is generally favorable,[32] though usually temporary; many radiologists feel that such therapy is only palliative. In the opinion of Cunning,[8] the sulfonamides are likewise of only temporary value. The potentially favorable or curative effect of streptomycin has been described by New, *et al.*[27]

Trauma of the trachea occurs infrequently in civilian life,[25] although many instances of such injury were observed in combat.[22,23] The resiliency, mobility and compressibility of the structure, its sheltered position and the surrounding cushion of soft tissues protect it from crushing injury. Lacerations, penetrating sounds and injury by striking the neck against a narrow-edged object are somewhat more common.

In severe lacerations involving the trachea, escape of air into the wound is obvious. In other forms of external trauma, subcutaneous or mediastinal emphysema, along with hemoptysis, may indicate rupture of the trachea. Spontaneous rupture of the trachea from violent coughing has been reported.[25] Wheezing and dyspnea depend on reduction of the tracheal lumen by submucosal hemorrhage and edema, or compression by hematoma external to the trachea.

Lacerated wounds involving the trachea may, if relatively clean, be closed with a minimum of drainage, providing injury

(a) (b)

Fig. IV. Scleroma of the larynx and trachea in a Russian-born male of 52 years. Dyspnea, hoarseness and wheezing of three years' duration. Diagnosis by tracheoscopic biopsy and bacteriologic study. Temporary improvement following deep Roentgen irradiation. (a) Planigram. (b) Lipiodol tracheogram.

to the trachea is not severe. Contaminated wounds, or those in which the tracheal wall is badly damaged, should be closed loosely to favor free drainage and a tracheal cannula left *in situ* for several days with adequate systemic biotherapy.

In the case of closed wounds, close observation for increasing dyspnea is the most important consideration. Asphyxia may be averted by introduction of the bronchoscope, followed by orderly tracheotomy, the cannula being left *in situ* until Roentgen examination, tracheoscopy and clinical trial by corking demonstrate an adequate airway.

The problem of treatment of cicatricial stenosis occurring as an end-result of perichondritis of the tracheal cartilages is discussed below. Cases in which stenosis occurs as a result of blast injury or exposure to noxious gases, such as those reported by Knight,[22] present a similar problem.

Chronic Cicatricial Tracheal Stenosis. Cicatricial narrowing of the trachea may be observed as a sequel to trauma, but more often, the condition represents the healed end-result of a chronic granulomatous lesion (tuberculosis, scleroma, lues, etc.). Cicatrization of a tuberculous lesion is the most common cause. Appreciable degrees of cicatricial narrowing at the site of tracheotomy are unusual, even when the cannula has been worn for prolonged periods, providing the tracheotomy has been performed at the proper level to avoid contact with the cricoid cartilage; however, persisting obstructive granuloma at the inner end of the tracheotomic fistula may be evident when decannulation is attempted, particularly in young children, and may require forceps removal.

Careful preliminary Roentgen and tracheoscopic study is a prerequisite to treatment; planigraphic films may be especially helpful. Keen,[19] in 1924, described an operation for cicatricial stenosis of the trachea, and recently Daniel[9] has demonstrated the ability of the trachea and larger bronchi to regenerate around hollow tubes of glass, plastic or inert metal. Taffel[37] has described the use of free fascia grafts in the repair of defects of the tracheal wall.

The more conservative treatment consists in either *1.* peroral dilatation, using smooth-tipped bronchoscopes in increasing sizes or metal bougies of the type used in treating laryngeal stenosis, or *2.* use of indwelling apparatus of rubber or plastic, if the stenosis is at such a level that a tracheal cannula

may be left *in situ* below the apparatus. Tracheostomy may be required to insert such apparatus initially; the "core-mold" may be anchored to the tracheal cannula or to a segment of rubber tubing lying on the skin surface; the latter method has the advantage of allowing daily changing of the tracheal cannula. Rubber dilators may be changed weekly, with increase in size until a No. 36 to No. 40F. in the average adult is tolerated for a period of three to four weeks. A test period of several months without apparatus is required before decannulation is attempted.

The technique of Erich,[11] in which excision of scar tissue and skin grafting are followed by the wearing of an acrylic mold, is likewise applicable to stenosis of the upper trachea and is said to accomplish restoration of an adequate tracheal lumen in a somewhat shorter period of time.

Peroral dilatation of a cicatricial tracheal stenosis cannot be accomplished rapidly. Progress must be slow, for excessive stretching is likely to be followed by a temporary increase in obstructive dyspnea, which may be a serious problem if a tracheal cannula is not being worn, and even more so when tracheotomy is not feasible, as in stenosis near the lower end of the trachea. In cases where impairment of bronchopulmonary drainage is evident, frequent bronchoscopic aspiration beyond the stenosis may be helpful.

Tumors of the Trachea. The relative infrequency of tracheal tumors is indicated by the statistics of various authors relating their incidence to that of laryngeal tumors. These ratios, as cited in the paper of D'Aunoy and Zoeller,[10] are 1:100 (Semon), 7:300 (von Bruns), 3:748 (Schmidt) and 4:800 (Mackenzie). D'Aunoy and Zoeller, in 1929, were able to collect 351 reported cases of primary tracheal tumors; in this group, benign and malignant tumors occurred with about equal frequency.

Benign papilloma is the most common of the truly neoplastic benign tumors, and fibroma is second in frequency. The coincidence of benign papilloma of the trachea and tracheal ectasia has been observed.[21] Other benign neoplasms less

frequently seen are adenoma, cylindroma, "mixed tumor," lipoma, angioma, chondroma and osteoma. The nature of the tumor must, of course, be determined by tracheoscopic biopsy. Benign tumor-like conditions, not truly neoplastic, are intratracheal goiter, amyloid tumor and tracheopathia osteoplastica (multiple osteochondromata).

Papilloma and fibroma are adequately treated by tracheoscopic removal, and the same is true of other benign neoplasms, except for those which are integral with the tracheal wall. In the case of vascular lesions, electrocoagulation is preferred to forceps removal. Pedunculated tumors may be removed with the snare.

The incidence of *malignant tumors of the trachea* as compared to that of adjacent portions of the respiratory tract, is surprisingly low. Culp,[7] in 1938, found only 82 reported cases of histologically proven primary carcinoma of the trachea, and Schoolman[34] found only five carcinomas of the trachea during a period in which 137 carcinomas of the larynx were seen. The relative frequency, as compared to the incidence of primary malignant bronchial tumors, is probably no greater than 2 per cent. As to sex, the male predominates in ratio of about 2:1.

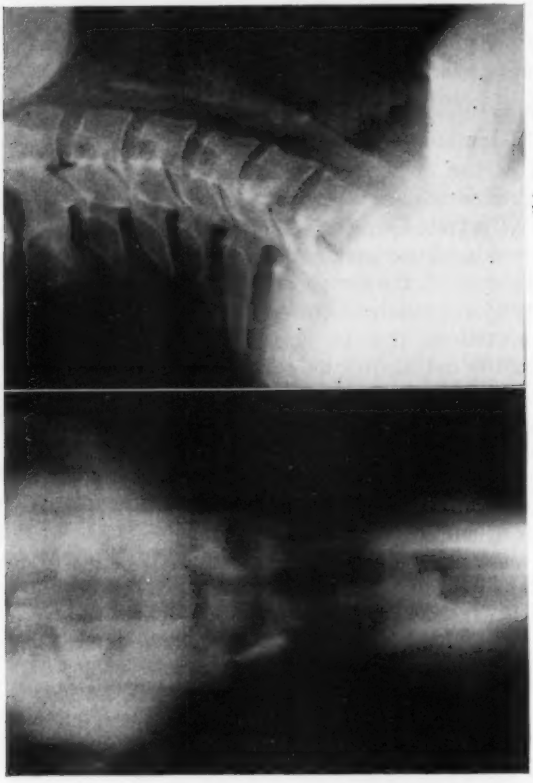
In somewhat more than half of the cases, the lower third of the trachea appears to be the site of origin. The posterior wall is said to be more frequently involved than the lateral wall and anterior wall. Squamous cell carcinoma and adenocarcinoma occur with about equal frequency. The "cylindroma" type of adenocarcinoma, usually of low-grade malignancy, is identified microscopically by the occurrence of columns of small cells of uniform size, with infrequent mitoses but some tendency for formation of acini containing mucus. This type of tumor must be differentiated from the benign adenoma and the so-called "mixed tumor." Microscopic appraisal of degree of malignancy, according to the criteria of Broders, allows some estimate of the rapidity with which local invasion and metastasis may be expected to occur. Metastases, more commonly seen in the squamous cell than in other histologic

types, are usually to the mediastinal, cervical or peribronchial nodes; distant metastases are unusual. Local invasion of adjacent structures is, of course, relatively common.

The most characteristic clinical manifestation of tracheal tumor is the "asthmatoid wheeze," which, in the early stages, is best heard at the open mouth near the end of a forced expiration, is increased by exertion, and may, if the tumor is pedunculated, be affected by change of position. The accompanying dyspnea is of increasing severity; paroxysmal exacerbations may occur if the lesion is pedunculated, but more often such episodes, usually nocturnal, accompany the accumulation of secretions due to obstructed tracheobronchial drainage. Cough is not always an early symptom, but paroxysmal cough and expectoration of purulent sputum occur when the increasing obstruction results in impaired drainage and secondary infection. Hemoptysis and blood-streaking are commonly observed. The initial symptoms may be those of esophageal invasion (dysphagia, regurgitation), or recurrent nerve paralysis (hoarseness).

The presence and location of the tracheal tumor may be well shown by the lateral Roentgenogram, or the planigraphic Roentgen film [see Fig. V (a)]. Films made following instillation of iodized oil may likewise demonstrate the lesion.

Tracheoscopy with biopsy under direct vision is the only procedure by which accurate diagnosis may be established. General anesthesia for this procedure should be avoided, particularly in the presence of obstructive dyspnea, and facilities for the endoscopic insufflation of oxygen should be available. The gross appearance of a malignant tracheal tumor varies to some degree with the histologic type; the squamous cell carcinomas of high malignancy present an ulcerated, nodular or fungating surface which may involve a large portion of the circumference of the lumen. Adenocarcinomas of low grade may be more localized to the point of origin, or even pedunculated, with a surface which is smooth or lobulated. The "cylindroma" type of adenocarcinoma may be covered with mucous membrane which is entirely smooth.



(a)

(b)

Fig. V. (a) Adenocarcinoma of the trachea in a colored female of 35 years. Increasing dyspnea, wheezing and productive cough of four years' duration. Diagnosis by tracheoscopic biopsy; treatment by forceps removal, electrocoagulation and deep Roentgen irradiation. Recurrence six years after treatment; death from pulmonary metastases nine years after initial treatment. (b) Adenocarcinoma of thyroid gland infiltrating trachea of a white male of 58 years. Increasing dyspnea and wheezing of about one year's duration. Diagnosis by tracheoscopic biopsy. No cervical node metastases palpable. Thyroidectomy and laryngotracheoectomy (Dr. W. Wayne Babcock); living without evidence of recurrence or metastasis after three years.

The differential diagnosis[16] includes endotracheal lesions of other types (benign tumor, granuloma, specific or nonspecific ulcerative lesions, cicatricial stenosis), as well as extratracheal lesions which may, by compression, narrow the tracheal lumen (aneurysm, esophageal tumor, or foreign body, lymphoblastoma or other space-taking mediastinal lesion). Other causes of wheezing respirations (bronchial asthma, bronchial foreign body, and partially obstructive lesions of the larger bronchi) may also require differentiation. Cases in which a tumor of bronchial origin appears to have extended proximally to

involve the trachea should not be classified with those of tracheal origin, although the difference is perhaps an academic one. Malignant tumors of the thyroid gland, with invasion of the trachea [see Fig. V (b)] are differentiated on the basis of histologic appearance. In a few instances where the "party wall" between the esophagus and trachea is involved, neoplastic tissue may be found in the lumen of both trachea and esophagus, so that the structure in which the tumor had its origin may be difficult to determine.

Complications of tracheal tumor include asphyxia, which may occur following a period of slowly increasing dyspnea; more commonly, accumulation of secretions will result in a number of of paroxysms of increased dyspnea, cough and cyanosis, during one of which asphyxia may occur abruptly; this is the usual terminal event. *Hemorrhage* is rarely fatal *per se,* but formation of obstructive blood clot may lead to asphyxia. *Bilateral obstructive emphysema* may be seen as a complication of tracheal tumor exhibiting the "ball-valve" action, and *atelectasis* of an entire lung may ensue if one of the main bronchi becomes occluded. Suppurative bronchitis, bronchiectasis and suppurative pneumonitis, the result of impaired drainage, may be observed. *Esophageal obstruction* or tracheoesophageal fistula, commonly followed by aspiration pneumonitis, may follow peritracheal invasion. Paralysis of the recurrent laryngeal or phrenic nerve, Horner's syndrome and superior caval obstruction are other manifestations of mediastinal invasion or metastasis.

Treatment of Malignant Tracheal Tumors. Prevention of asphyxia may be the first consideration. This may be accomplished by tracheoscopic removal of obstructing tumor tissue, if this can be done without excessive trauma or hemorrhage; if the tumor is a vascular one, electrocoagulation is preferred. Aspiration of secretions beyond the point of narrowing may facilitate drainage; however, attempts at vigorous dilatation of a tracheal lumen narrowed by neoplastic tissue should be avoided, for such manipulation may be followed by a rather abrupt increase in dyspnea. Tracheotomy for relief of dyspnea is feasible only if the obstruction is at a high level. Some

relief of temporary nature may accompany administration of oxygen or oxygen-helium mixture, preferably by mask.

Suppurative bronchopulmonary infection will require, in addition to periodic bronchoscopic aspiration, use of sulfonamides, penicillin or streptomycin, depending upon the susceptibility of the organisms cultured from the tracheobronchial secretions. In instances where esophageal obstruction has occurred, as a result of invasion or compression, gastrostomy may be required for feeding; gastrostomy is also indicated if a tracheoesophageal fistula is present, and an effort should be made to improve oral hygiene.

Complete extirpation of a malignant tumor by endoscopic removal is usually not possible, although Broyles[5] has reported a case of squamous cell carcinoma surviving 11 years after treatment by fulguration. Curative treatment by surgical resection is possible only if the lesion is limited to the upper third of the trachea. In a few instances, if the tumor is pedunculated or well localized, removal by tracheofissure, with excision or electrocoagulation, may be feasible. More extensive lesions will require resection of a portion of the upper trachea and adjacent structures; partial or total laryngectomy may be necessary. Presence of cervical node metastases calls for complete neck dissection. The likelihood of recurrence is somewhat diminished by implantation of radon seeds at the time of operation, but the potency and distribution of the seeds should be such that necrosis of remaining cartilage will not occur.

The prognosis in carcinoma of the trachea is generally unfavorable. Lesions of the upper trachea are more amenable to either surgical treatment or irradiation, and offer a somewhat better prognosis than those at lower levels. The adenocarcinomas of low grade appear to be the most favorable histologic type.

Tracheal Compression. The clinical manifestations of partial tracheal obstruction due to compression are essentially the same as those of intrinsic or intratracheal obstruction. Wheezing and dyspnea are predominantly expiratory, though

less so when the cervical trachea is compressed; the characteristic "brassy" cough idicative of compression of the thoracic trachea is well known. Other symptoms, signs and Roentgen findings are those of the pathology responsible for the compression.

Compression by hematoma, with dyspnea, may occur following injury or following operations on the neck. Since thyroidectomy is one of the more common operations prone to be followed by this complication, it is important to differentiate dyspnea due to tracheal compression from that due to bilateral injury of the recurrent laryngeal nerves. The latter condition is characterized by inspiratory dypsnea, inspiratory stridor and impairment or loss of voice in the initial stages; indirect laryngoscopy will show the vocal cords motionless in the midline, and tracheotomy will be required in many instances.

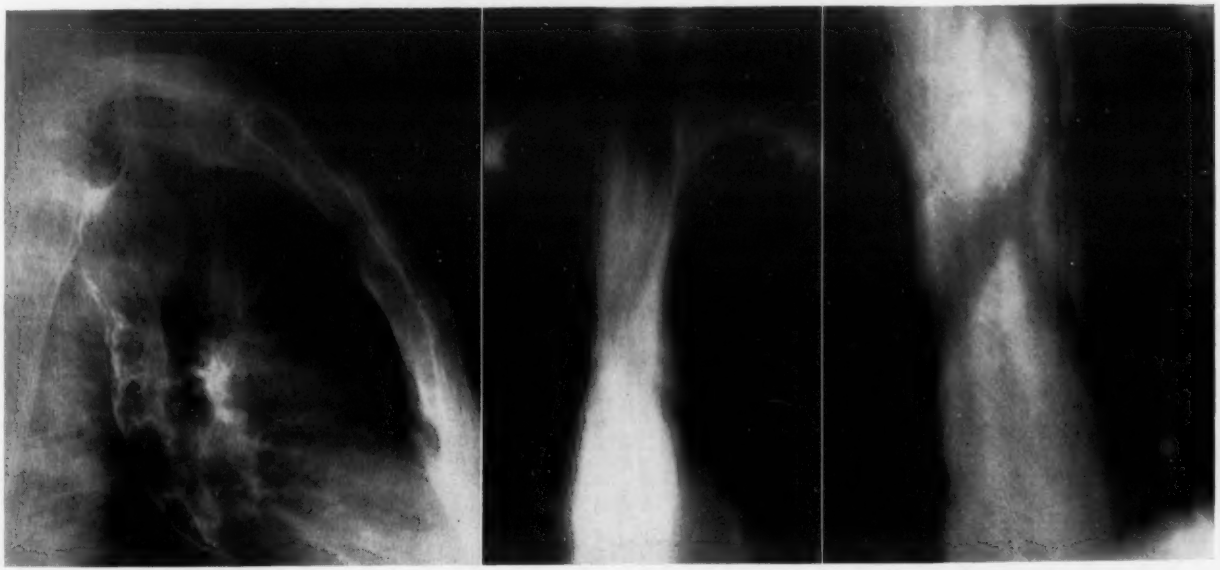
Differentiation of compression by hematoma following trauma from obstruction due to injury of the trachea itself may be difficult, although occurrence of hemoptysis and presence of interstitial emphysema speak for the latter. The indications for treatment, if dyspnea is marked, are the same in these conditions as in postoperative hematoma. A bronchoscope or long endotracheal catheter should be introduced and the trachea exposed by external incision (or reopening of the incision in the postoperative cases). Arrest of bleeding and evacuation of hematoma if present will relieve the compression. If the tracheal wall has suffered considerable injury, a tracheal cannula may be inserted.

Compression due to acute infection may be observed in periesophageal cellulitis, retroesophageal abscess or mediastinitis due to foreign body or instrumental perforation of the hypopharynx or cervical esophagus. Roentgen study is extremely helpful;[20] the lateral film [see Fig. III (b)] is of particular value in demonstrating the degree of widening of the retrotracheal soft-tissue zone and the tracheal compression. Presence of interstitial air is nearly always conclusive evidence of perforation; since knowledge of the exact site of perforation

is not essential, the administration of barium is avoided, unless necessary for localization of perforating nonopaque foreign bodies.

Although some cases of mediastinitis due to foreign body or instrumental perforation may respond to biotherapy (especially if the perforation is large enough to allow adequate internal drainage) the occurrence of tracheal dyspnea is an indication for surgical drainage; this is usually accomplished by cervical mediastinotomy, following preliminary insertion of a bronchoscope or endotracheal catheter, the prevertebral space being opened through the interval between the sternomastoid muscle and carotid sheath, laterally, and the thyroid gland, trachea and esophagus medially. If, following drainage, dyspnea recurs when the bronchoscope or endotracheal catheter is withdrawn to the glottic level, a tracheal cannula should be introduced through a separate incision. Intensive biotherapy, controlled by culture and susceptibility tests, and attention to oral hygiene are indicated; saliva should be expectorated rather than swallowed. Usually a feeding tube can be introduced if this is done under fluoroscopic guidance with care; otherwise, parenteral feeding is required temporarily.

Tracheal compression due to *enlarged thymus*, and that due to *esophageal foreign body*, have been mentioned above. Other types of compression, developing more slowly, are those of substernal goiter, thyroid carcinoma or abscess of the thyroid gland, aneurysm of the aorta or subclavian artery [see Fig. VI (b)], benign tumor of the upper mediastinum, mediastinal cyst, invasive tumor of bronchogenic [see Fig. VI (c)] or esophageal [see Fig. VI (a)] origin, and mediastinal lymphadenopathy (inflammatory, metastatic tumor, lymphoblastoma). Heuer[13] found primary lymphoblastoma and metastatic lymphadenopathy of about equal frequency; most of the cases of the latter represent extension from a bronchogenic carcinoma. Differential diagnosis will require, in addition to careful general examination and indirect laryngoscopy, Roentgen examination of the neck, chest and esophagus, and may require *1.* biopsy of any enlarged cervical nodes, *2.* bronchoscopy or esophagoscopy or both, except in cases where aneurysm is a



(a) (b) (c)

Fig. VI. (a) Tracheal compression by carcinoma of esoph-agus in a white male of 69 years. (b) Tracheal compression by luetic aneurysm of the subclavian artery in a white male of 40 years. (c) Tracheal compression by bronchogenic carcinoma infiltrating mediastinum.

likely possibility, *3.* planigraphic Roentgen examination, and *4.* possibly needle aspiration biopsy under fluroroscopic guidance. Laboratory procedures which may be relevant include serologic study for lues, differential blood count with particular attention to young forms, sternal marrow biopsy, cytologic study of bronchial secretions and basal metabolism.

SUMMARY.

1. General characteristics of the phenomenon of tracheal obstruction are discussed.

2. Diagnostic features of the various types of tracheal obstruction are described and their treatment indicated.

3. Illustrative cases are presented.

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DR. SEYMOUR H. RINZLER, NEW YORK UNIVERSITY, WINS 1949 MISSISSIPPI VALLEY MEDICAL SOCIETY ESSAY CONTEST.

Seymour H. Rinzler, B.A., M.D., F.A.C.P. Adjunct in Medicine and Cardiology, Beth Israel Hospital, and Instructor in Rehabilitation, New York University, is the winner of the ninth annual essay contest of the Mississippi Valley Medical Society "for the best unpublished essay on a subject of practical and applicable value to the general practioner of medicine." Dr. Rinzler's paper is entitled, "Present Status of Medical and Surgical Therapy in Angina Pectoris." Second prize goes to Dr. Edward D. Robbins, of the University of Chicago, for his essay, "Hypometabolism or Hypothyroidism," and third prize to Dr. Wallis L. Craddock, Ft. Logan, Colo., for his paper, "The Problem of Histoplasmosis."

Dr. Rinzler will receive a cash award, a gold medal, a certificate of award, and will present his essay at the Fourteenth Annual Meeting, Mississippi Valley Medical Society, Jefferson Hotel, St. Louis, Sept. 28, 29, 30. His paper and those of Dr. Robbins and Dr. Craddock will appear in the January, 1950, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.).

TREATMENT OF CARCINOMA OF THE LARYNX: A STATISTICAL STUDY OF RESULTS.*

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The statistical method measures the efficiency of different treatments for the same disease. The wider the source of the statistics in number of cases, in time and area, the more accurately they reflect the general rule. Carcinoma of the larynx has large variations in symptoms, malignancy, treatment and prognosis. New operations and irradiation under their respective indications as the method of choice have been increasingly suggested during the past decade as either equal or superior in some respects to those usually employed. Consequently, a statistical study on a broad factual basis of the results of therapy may be of value in establishing the general rule for the respective indications of the several treatments as well as their prognosis.

The source of the statistics are the reports on the results of treatment for carcinoma of the larynx for a five-year period, which have appeared in the American and English medical literature during the past 10 years. Many reports, otherwise excellent, are not included because the five-year results could not be accurately identified. The series has a total number of 4,584 cases, of which 2,213 have a report on some phase of five-year results, and 2,371 are untraced or less than five years. The series is based upon the reports of — omitting joint authors—Arbuckle,[1] Bernheimer,[2] Brunschwig,[3] Clerf,[4] Colledge,[5] Coutard,[6] Cunning,[7] Cutler,[8] Desjardins,[9]

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Hare,[10] Howes,[11] C. L. Jackson,[12] Kernan,[13] LeJeune,[14] Lentz,[15] Lederman,[16] McCall,[17] Negus,[18] New,[19] Orton,[20] Schall,[21] Tucker,[22] and my own small group. Acknowledgment is made of the free use of the material cited above. The reports follow no fixed pattern; so that the number of cases reported on the results of different types of treatment has wide variations, some being large and others relatively small. The consolidation of these results has been made to compare the relative efficiency of the several methods of treatment.

The type of treatment for carcinoma of the larynx is largely determined by the age, the physical condition of the patient, the site, size and degree of malignancy of the tumor, and the motility of the vocal cords. Fig. 1 shows the age incidence in percentage by decades. The curve of incidence demonstrates that laryngeal cancer covers the span of life from the early "teens" to 90 years and beyond these limits will very rarely be found. The physical condition of a patient occasionally determines whether definitive treatment can be undertaken. Patients with chronic pulmonary tuberculosis, diabetes and cardiovascular disease are poor risks for either surgery or irradiation. The site of the initial lesion occurs in the order of frequency on the vocal cord, supraglottic, subglottic and extrinsic areas. In cancer clinics, however, the neoplasm may occur more often in the supraglottic area, *i.e.*, epiglottis, aryepiglottic fold, ventricular band and in the ventricle, than on the vocal cord. The symptoms are dependent, of course, on the location and extent of the tumor. Cancer of the vocal cord has hoarseness as an early symptom; but elsewhere there are no early symptoms. The first indication of a subglottic, supraglottic or extrinsic cancer may be one or the other of dyspnea, odynophagia, dysphagia, hemoptysis and metastasis to the cervical lymph nodes. Pain in the throat or the ear is a late symptom.

Diagnosis is the beginning of treatment. A biopsy is primarily for differential diagnosis between cancer and keratosis, papilloma, granulations, tuberculosis, syphilis and monilliasis; and secondarily for estimating the degree of malignancy.

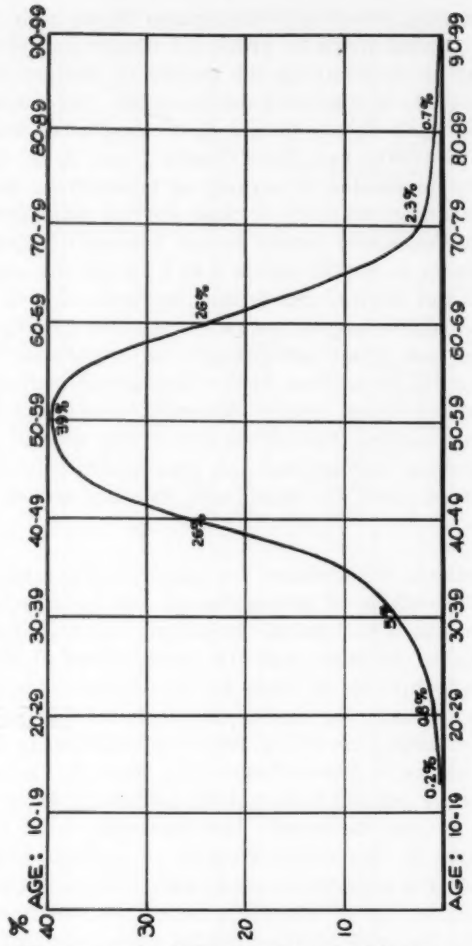

Fig. 1. Age incidence of carcinoma of larynx.

Keratosis and recurrent papilloma in the adult are wolves in sheep's clothing and are assumed to be malignant until contrarily proven. The biopsy is essentially a sampling process and subject to the inherent advantages and weaknesses of

such a method. For this reason, it may be necessary to repeat a biopsy several times to prove the diagnosis. The value of the biopsy in determining the degree of malignancy and of radiosensitivity is at present controversial. The consensus now is that Broder's Grades 3 and 4, or anaplastic cancer, have a higher mortality rate than Grades 1 and 2, or the epidermoid, whether treated by surgery or irradiation; that Grades 1 and 2 in a large majority of cases are not radioresistant, and that a radiosensitive cancer is not necessarily radiocurable. Cordal cancer is usually Grade 1 or 2; while the supraglottic, subglottic and extrinsic are frequently Grades 3 or 4. Metastasis from cordal lesions occurs late, due to the paucity of lymph vessels and low grade malignancy; but metastasis from elsewhere is early on account of the high grade malignancy and rich supply of lymph vessels. The prognosis is not dependent on any one factor. The initial site of the tumor, its extent on the mucosal surface and the penetration into the underlying tissues must be considered in conjunction with the grading.

The methods of treatment for cancer of the larynx are the classical operations of laryngofissure and laryngectomy, special operations with limited indications, fractionated irradiation (Coutard method) and the combination of these. The respective techniques of these are an evolution from the contributions of many laryngologists, surgeons and radiologists, and, in the main, are today rather standardized. The anesthesia of choice in laryngofissure was local; in laryngectomy, either local or general from sodium pentothal. My experience, for what it may be worth, has been that local anesthesia accompanied by the administration of sodium pentothal in laryngectomy is superior to either alone. Since the prevention of infection by the prophylactic use of the sulfonamides and antibiotics, the postoperative deaths were practically limited to coronary thrombosis and apoplexy.

Intralaryngeal surgery, as now defined, was adapted by Lynch and developed by LeJeune and New. It is a special operation with indications limited to a lesion on the mid-third

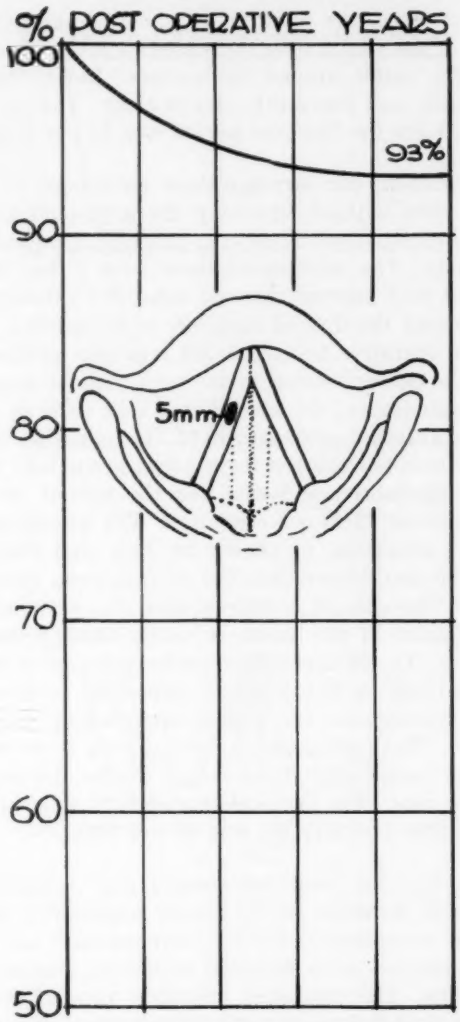

Fig. 2. A graph for the survival rate for intralaryngeal surgery. (The survival curve in this and following graphs is schematic for the first, second, third and fourth postoperative years.)

of the vocal cord, the motility of which is unimpaired. The long axis of the tumor does not exceed 5 mm., with the vocal cord clearly visible around its borders. Later, the voice is slightly husky and frequently almost clear. The survival rate (see Fig. 2) for the five-year period was 93 per cent.

The indications for laryngofissure have been a cancer on one vocal cord without involving the arytenoid and, where occurring, an extension across the anterior commissure to the opposite side. The contraindications were either fixation of the affected cord, supraglottic and subglottic extensions, penetration through the thyroid cartilage or metastasis. The indications for operative technique fell into two groups: In the first was the cancer limited to one cord without involving the anterior commissure; the second was that with an extension across the anterior commissure to the opposite cord. The surgical technique employed for the first group may be termed cordal laryngofissure, and that for the second, commissure laryngofissure or Jackson's operation. The submucosal extension of the neoplasm, as shown by New and Fletcher,[23] is from 5 to 15 mm. larger than the surface area, dependent on the grade. The rule of 1 cm. between the excision and the apparent border of the lesion is nearly always safe, except for Grade. 4. The factors of the submerged cancer and of the tendon described by Broyles[24] are important in selecting the type of laryngofissure for lesions approaching the anterior commissure. The postoperative deaths were 2 per cent. The deaths from causes other than cancer for the five-year period were 10 per cent. The survival rate of laryngofissure for the five-year period (see Fig. 3) was 76 per cent.

The tendency for local recurrences and metastasis after laryngofissure increases as the cancer approaches or crosses the anterior commissure. Cordal laryngofissure and commissure laryngofissure were reported under the general term of laryngofissure. The combined mortality rate after the two operations, as stated above, was 24 per cent. What part of this rate followed the one operation or the other? The available evidence on this question was studied and an estimate

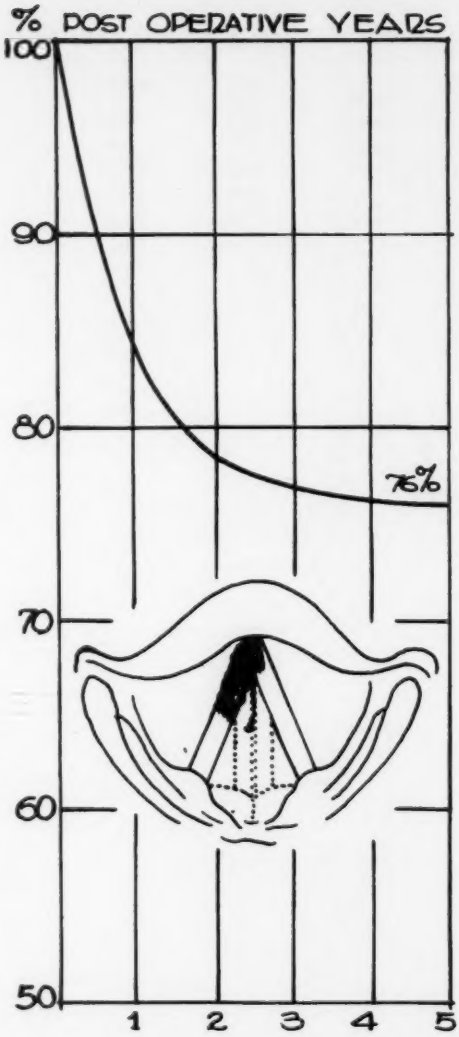

Fig. 3. A graph for the survival rate of laryngofissure.

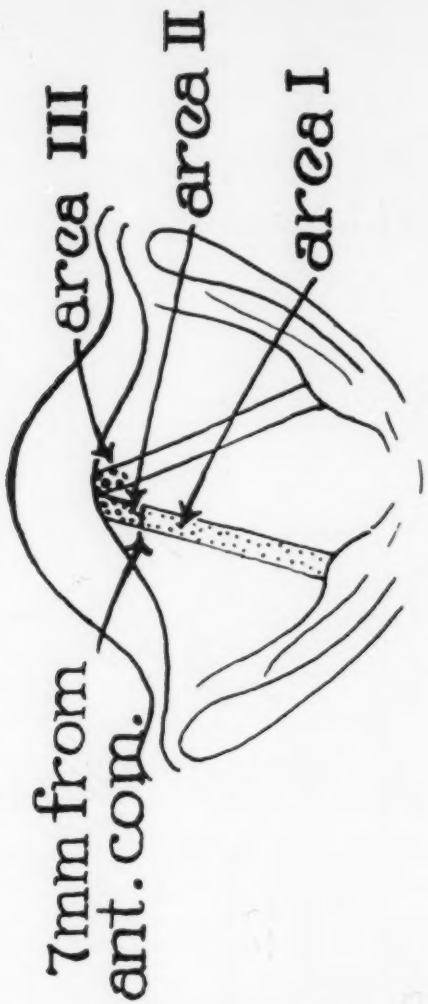

Fig. 4. Extent of cancer for cordal laryngofissure, Area I; for commissure laryngofissure, Areas I and II; for laryngectomy, Areas I, II and III.

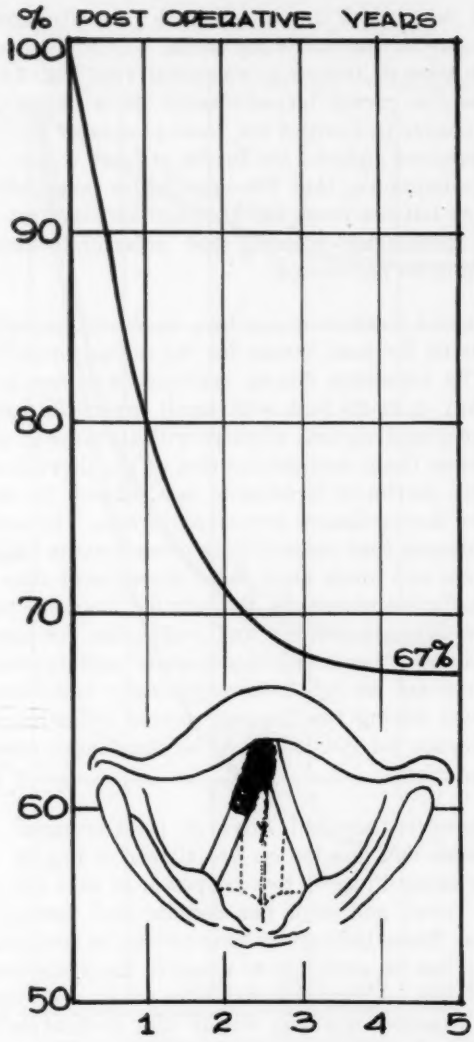

Fig. 5. A graph for the survival rate of irradiation for indications of laryngofissure.

made that the ratio of the cancer death rate after commissure laryngofissure to that following cordal laryngofissure was two to one. In view of this, it is suggested (see Fig. 4) that the indications for cordal laryngofissure be a cancer with its posterior border in front of the vocal process of the arytenoid and its apparent anterior border be at least 7 mm. from the anterior commissure; that for commissure laryngofissure the lesion reach but not cross the anterior commissure; and that for those neoplasms crossing the anterior commissure a laryngectomy be performed.

Fractionated irradiation has been used with increasing frequency during the past decade for the indications of laryngofissure. The consensus among radiologists seems to be that radiotherapy is at its best with small superficial cancer, but becomes progressively less effective with increase in size, invasion of deeper tissue and penetration of the thyroid cartilage. The relative merits of irradiation and surgery in cordal and commissure laryngofissure are controversial. The evidence at present indicates that surgery for a cordal cancer has a higher survival rate and much more voice impairment than irradiation, but sufficient statistical data are not available to make a comparison between surgery and irradiation for commissure cancer. Radiotherapy is still experimental and its precise indications have not as yet been statistically established. The survival rate during the five-year period following fractionated irradiation for indications of laryngofissure (see Fig. 5) was 67 per cent.

The respective survival rates of intralaryngeal surgery, laryngofissure and irradiation are shown in Fig. 6. Each of these is designed to serve two purposes, to save life and preserve the voice; and each has definite and sharply limited indications. These indications have proven to be rigid and not elastic, and can be stretched to preserve the voice only at the expense of life. A laryngologist may occasionally break the rules and preserve a voice; but if this is done in a series of cases, his patients pay the hard price of an increased mortality rate.

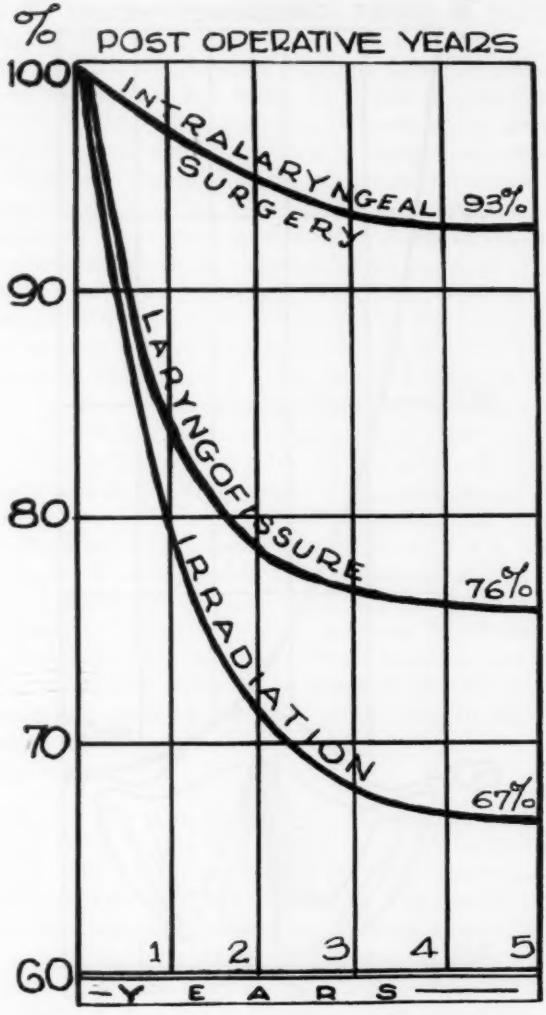

Fig. 6. A graph for comparison of respective survival rates.

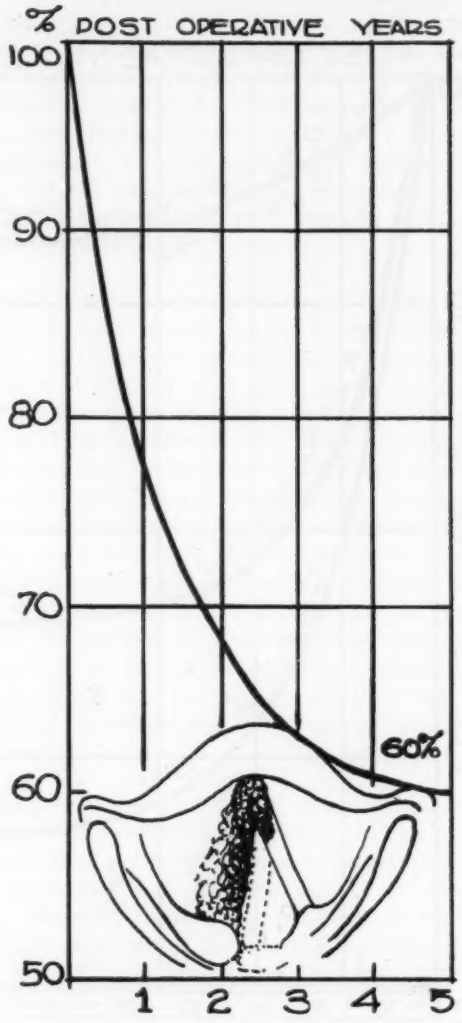

Fig. 7. A graph for survival rate after laryngectomy.

The indications for laryngectomy are either carcinoma involving all the superior surface of a vocal cord, fixation of the cord, invasion of the cartilage, extension to the other cord, to the supraglottic, subglottic or extrinsic areas, or metastasis to the regional nodes. The consensus is that advanced age of the patient, poor physical condition, original site of neoplasm in supraglottic area with invasion of hypopharynx and metastasis, widespread extension from cordal cancer to extrinsic area, hypopharynx or base of tongue and bilateral metastasis are considered to be contraindications. Some laryngologists regard a metastasis to the cervical lymph nodes at the first examination and others consider a large anaplastic neoplasm in the supraglottic area as contraindications for laryngectomy. McCall's method for preoperative voice training has received endorsements. Postoperative training for developing an esophageal voice was held obligatory. No report was made on the technique for closure of the cricopharyngeus muscle to facilitate acquiring an esophageal voice. Postoperative deaths were 3 per cent, and deaths from causes other than cancer during the five-year period were 10 per cent. The survival rate during the five-year period for laryngectomy (see Fig. 7) was 60 per cent. It is estimated that postoperative metastasis occurred in about 30 per cent of the laryngectomies without the preoperative indication of their presence. Block dissection of the cervical region was considered an essential part of the operation in several reports, as prophylactic postoperative irradiation had proven to be of doubtful value in this group of cases, except probably those with the initial lesion in the supraglottic area.

Resection of the thyroid cartilage followed by irradiation as adapted by Arbuckle is a special operation with limited indications. The indications for the operation are those cases "ordinarily considered" to be inoperable. The normal thyroid cartilage is as tolerant to irradiation as other tissues; but the tolerance is lowered by infection, invasion of cancer, partial resection and previous irradiation. The resection of the thyroid cartilage in any event prevents its radionecrosis. The survival rate during the five-year period for resection of the thyroid cartilage and irradiation (see Fig. 8) was 38 per cent.

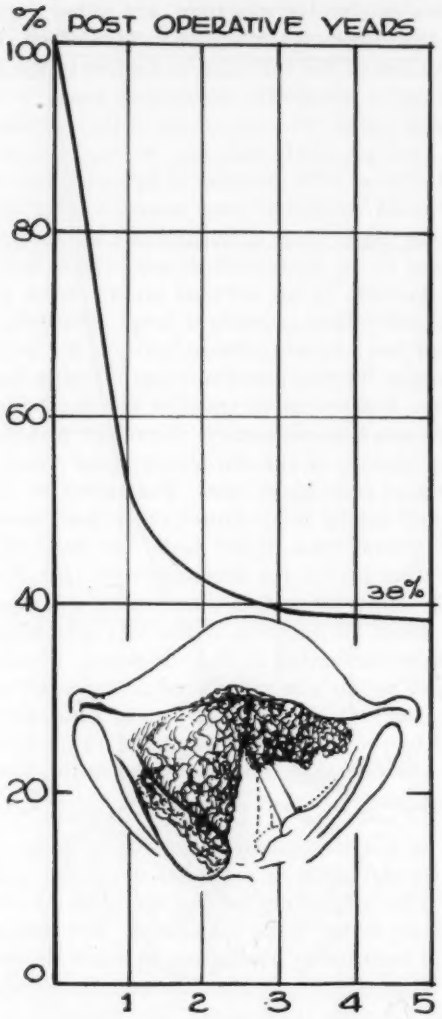

Fig. 8. A graph of survival rate for resection of the thyroid cartilage and irradiation.

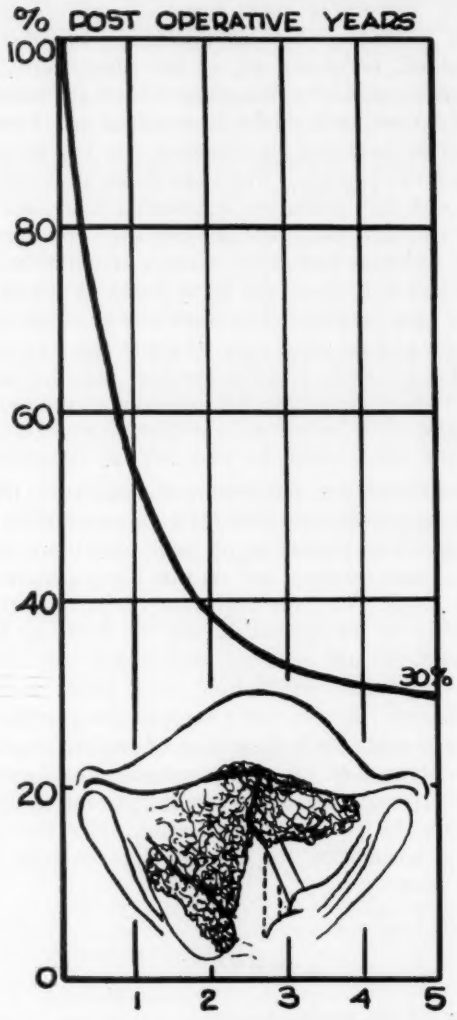

Fig. 9. A graph of survival rate after irradiation for salvage therapy.

In the reported statistics on fractionated irradiation of the larynx, the inoperable cases were not differentiated from those with indications for laryngectomy; however, it is assumed that all, or nearly all, of the cases were inoperable. The consensus among radiologists was that fixation of the vocal cord did not contraindicate radiotherapy. Irradiation of the inoperable cases may be classified into two groups, palliative and salvage therapy. The unanimous statement of those reporting was that palliative irradiation increased suffering, shortened life and was contraindicated. The survival rate during the five-year period for salvage irradiation on inoperable cases (see Fig. 9), to the great credit of the radiologists, was 30 per cent. Incidentally, Coutard's survival rate during five years for similar cases some 20 years ago was 27 per cent. Not included in above was the survival rate during the five-year period after irradiation for extension to the hypopharynx of 7 per cent.

Local recurrence was reported more frequently than metastasis after laryngofissure; but after laryngectomy, the ratio was reversed. The treatment of local recurrence and metastasis was salvage therapy, and on this basis gave an excellent account of itself. The survival rate of salvage therapy by either surgery or irradiation during the five-year period for local recurrences and regional metastases was as follows: Laryngofissure, after irradiation, very poor; laryngectomy after irradiation, 50 per cent; laryngectomy after laryngofissure, 39 per cent; block dissection of regional metastasis of those cases treated at second operation after laryngectomy, 44 per cent; irradiation after irradiation for indications of laryngofissure, 32 per cent; irradiation after laryngofissure, 35 per cent; irradiation for local recurrence after laryngectomy, very poor.

SUMMARY.

1. Cancer of the larynx has wide variations in symptoms, malignancy, treatment and prognosis.

2. The biopsy is valuable in differential diagnosis and to define the degree of malignancy; but not in estimating radiosensitivity.

3. Broder's Grades 1 and 2 have a higher survival rate than Grades 3 and 4 after both surgery and irradiation.

4. Intralaryngeal surgery has the highest survival rate and best functional result due to early diagnosis.

5. The present indications for laryngofissure are satisfactory if preservation of a useful voice is considered to be more important than saving life; but if saving life be more important, a revision of the indications is indicated.

6. Irradiation has a lower survival rate and much better voice than surgery for the indications of laryngofissure.

7. Routine block dissection is indicated with laryngectomy, for approximately 30 per cent of these cases have masked metastases.

8. The survival rate of resection of the thyroid cartilage with irradiation on inoperable cases suggests its further trial in salvage therapy.

9. These statistics indicate that surgery and irradiation are complementary and not competitive. The survival rate of 30 per cent for irradiation in salvage therapy saves many lives otherwise lost.

10. The triad for treatment is early diagnosis, saving life and preservation of a useful voice. Of these, early diagnosis is the most important, for on it the other two are dependent.

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THE DYNAMICS OF NASAL MUCUS.*

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One of the most universal complaints of mankind is post-nasal discharge. Although disagreeable to the patient, it is also one of the most annoying conditions to treat in nose and throat practice. The function of any mucous membrane is to secrete mucus and this should be constantly kept in mind and impressed on the patient.

From the time of Hippocrates and for the following 2,000 years, the brain was considered the origin of mucus which reached the nose through the cribriform plate and spread over the nasal membrane. Quoting from Wright,[1] "When we realize that the ancients, Hippocrates (460 B. C. — 375 B. C.), Galen (130 A. D. — 200 A. D.) and their followers knew nothing of the muciparous glands, and, of course, nothing of the function of these microscopic structures, it is easy to understand the absolute mental necessity for them to find some explanation of the origin of the secretions which bathe not only the respiratory tract but the gastrointestinal mucous membranes as well. As for the moisture of the lungs, it is natural that they should look for some explanation in the liquids swallowed. This lack of knowledge, as well as a mistaken anatomical observation, led them into another error which persisted still longer. The cribriform plate of the ethmoid bone (the sieve-like bone) at the top of the respiratory tract was usually seen only in the dried specimen by the ancients, unfamiliar with dissection of the human body. The

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idea arose that the humors were distilled in the gland-like contents of the cerebral cavities and sifted through the cullender plate of the ethmoid to parts below." This conception of the cerebral origin of nasal fluid was also shared by Galen, who lived 500 years after Hippocrates. In Galen's description of the nasal passages he said, "the mucus, especially such as is viscid, . . . is blown forcibly from the nostrils and is hawked out through the mouth."

People in all walks of life complain of catarrh or postnasal discharge, especially those who spend most of their time indoors. Individuals who depend on their voice in their vocation, such as public speakers, singers, clergymen, etc., often find it a trying affliction.

In determining the cause of this complaint, a detailed history is indispensable. The character of the discharge, whether watery or mucoid, the time of day, frequency, previous sinus infections, type of work, whether indoors or outdoors, personal habits, including smoking and alcohol, diet, rest, general health, exercise, mental strain, emotional upsets, etc., should all be carefully noted. Many of these conditions can be corrected or avoided, while others, such as environment, are beyond our control.

With these facts in mind, let us consider a few points about normal mucus. In a healthy nose, mucus is composed of about 2 to 3 per cent mucin, 1 to 2 per cent salts (sodium, calcium and potassium) and about 97 per cent water. This proportion differs slightly in individuals and under various climatic conditions. For instance, nasal mucus tends to be more viscid in a temperate climate than in a warm, humid atmosphere. This is partly due to the drying effect of artificial heat on the nasal mucous membrane. The active ingredient of mucus is mucin — a protein combined with glucose. It also contains a very complex polysaccharid closely associated with a protein. Mucins vary in their composition; "their solubilities and degrees of viscosity differ, the difference lying probably in the protein portion of the molecule."[2]

Nasal mucus differs in its essential characteristics from mucus obtained from any other source. Among other things, it has a much greater viscosity than the corresponding gastric mucin. Secretion from the nose containing 1.26 per cent protein has a viscosity equivalent to an 8.4 per cent secretion of gastric mucin.[3] In other words, nasal mucus is six to seven times as viscid as mucin from the stomach.

The whole mucosal lining of the nose from the vestibule to the nasopharynx (just below the level of the Eustachian tube), including the sinuses, secretes mucus to a varying degree. The serous and goblet cells of the posterior two-thirds of the septum and lateral wall, particularly in the meatuses in the region of the ostia, form an abundant amount of mucus and drainage in this area is very active. The stroma of the inferior turbinates is made up of large cavernous spaces with relatively few glands, while the middle and superior turbinates contain many glands and few cavernous spaces.

In general, the direction of the flow of mucus is downward and backward. From the frontal sinus, antrum and anterior ethmoid cells mucus is moved along by the cilia into the middle meatus and posteriorly along the lateral wall. It moves under the posterior end of the middle turbinate and then downward behind the soft palate and in front of the Eustachian tube just medial to the posterior pillar of the tonsil. A little bathes the surface of the tonsil, but the main stream passes down to the pyriform sinus, where it is either coughed up or swallowed. From the posterior ethmoid cells mucus streams down from the superior meatus in front of the Eustachian tube and joins the former stream from the anterior group of cells, at about the level of the tube.

A smaller stream from the posterior ethmoids goes behind the Eustachian cushion and joins the former combined streams at about the level of the soft palate. The stream of mucus from the sphenoid sinus tracks down the anterior wall from the ostium and fans out high up in the nasopharynx and well behind the Eustachian tube to join the other streams just below the level of the soft palate. These combined streams of

mucus follow down the posterior pharyngeal wall over the lateral band just medial to the posterior pillar.[4] If the mucus is infected, a lateral pharyngitis may result and if the cause is not eliminated a chronic condition is liable to occur. This (lateral pharyngitis) further increases the postnasal discharge

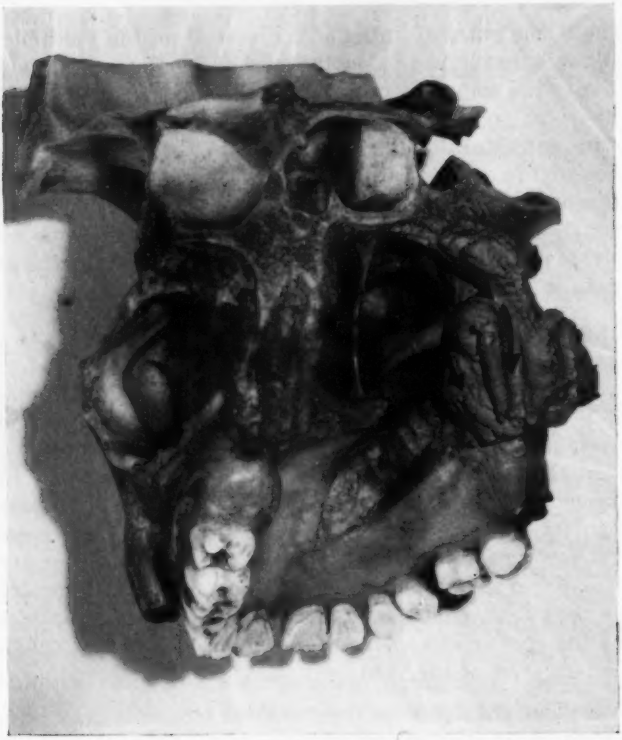

Fig. 1. Dried specimen to show the ciliary streams from the sinuses — down in front and behind the Eustachian cushion. In this way the streaming avoids the orifice of the tube.

In the normal nose a thin sheet of mucus is spread thinly over the whole membranous lining, including the sinuses, and extends downward through the esophagus to the stomach. It

also covers the mucosa of the larynx, the trachea, bronchi and even the smallest bronchioles. Mucus is colorless, elastic and slightly sticky and can be removed by suction in long strands before it breaks. This traveling belt is being constantly replaced and carries away foreign particles, such as dust and organisms which lodge on the surface. It is moved by the action of the cilia aided by traction, gravity and the act of swallowing. In the active posterior two-thirds of the nose, including the lateral wall and septum, normal mucus moves at the rate of 4 to 6 mm. per minute. Over the inactive area of the lateral wall and septum, including the anterior ends of the middle and inferior turbinates, normal mucus moves slowly into the meatuses — only a few millimeters per hour. In other words, there is a new layer of mucus about once every 10 minutes over the posterior two-thirds of the nose and a new film of mucus about once every hour or two over the anterior one-third. These observations, first described by Hilding[5] in 1932, are well known and have stood the test of time. Perhaps the manner in which this drainage occurs is not clearly appreciated. During the past winter, we have used a phase contrast microscope at McGill University in studying nasal mucous membrane. By means of this newer instrument greater detail and definition are obtained than by the standard type of microscope and, as the name implies, better contrast.

Pieces of mucous membrane from the medial wall of the middle and inferior turbinates show a definite pattern of ciliary streaming. Looking down on the surface and viewing the cilia from above, minute rivulets can be seen which course downward and join others to form larger streams. At times these streams are diverted as clumps of cilia move while others rest, but in general the streams tend to converge toward the meatuses.

They resemble streams which course down the mountains in the Spring, gaining momentum as they reach the sea. Instead of branches, logs and tree trunks — inhaled particles, dust and epithelial debris can easily be seen. In the same way this streaming effect is shown when small fragments of antral

mucosa are examined under the phase contrast microscope. The currents converge upward toward the ostium when the slide is perpendicular, *i.e.*, against the force of gravity. It is necessary, of course, to moisten the fresh tissue with Ringer-Locke's solution before applying the cover slip, but even when normal saline is used the ciliary beat is well maintained often for hours.

Ciliary movement is of necessity influenced by the consistency of the overlying blanket of mucus. If the proportion of mucin is increased so that the layer of mucus is thick and sticky, movement of the cilia is slowed down (even the addition of one-half of 1 per cent mucin to the normal 3 per cent increases the viscosity of nasal mucus tremendously). On the other hand, where there is an excess of watery mucus containing very little mucin, as in vasomotor rhinitis or hay fever, ciliary action is also impaired. It should be kept in mind that tremendous quantities of thin mucoid secretion flow down in allergic patients without bothering them, but often low grade allergies cause thickening of the mucus with resultant discomfort and postnasal discharge.

In an allergic nose without an excess of aqueous mucus, movement of the cilia on the water-logged tissue is not impaired. It appears to act the same as normal mucous membrane; however, any mechanical obstruction which interferes with normal air currents, causing dryness with stagnation of mucus is a potential source of postnasal discharge. Septal deflections, ridges, spurs, polypi, etc., in the anterior third of the nose are subjected to blasts of inspired air, and the exposed surfaces revert to a squamous type of epithelium without cilia. The drying effect makes the mucus viscid, and it lodges at the back of the nose.

Septal perforations anteriorly, whether due to disease or operative procedures are liable to cause dryness. Cross currents of inspired air increase the consistency of nasal secretions, and they tend to collect posteriorly.

Perforations in the posterior two-thirds of the septum, such as occur occasionally following submucous resection, as a rule,

cause little or no discomfort to the patients. This area deep in the nose is protected and the active ciliary movement carries the mucus away quickly.

Smoking and alcohol in moderation produce a mild congestion and hyperemia which in the early stages does not retard ciliary action. In chronic cases fixed tissue changes occur with a permanent dilatation of the blood vessels of the nose, and the mucus becomes thick and sticky. The overburdened cilia have difficulty functioning under these conditions and the constant clearing of the throat adds to the patient's discomfort. Unless there are pathogenic organisms present in the discharge, and this is infrequent, no harm results.

When infection is present as in acute sinusitis, postnasal drainage is a common complaint. This is, however, of a temporary nature and tends to disappear when the acute condition subsides.

In chronic cases the origin of the discharge should be investigated and if possible eliminated. Pus draining posteriorly is a constant source of annoyance and these patients do their best to remove it before it enters and "poisons the system." The ethmoids and sphenoids should be carefully examined and abnormal hypertrophies, especially of the posterior tips of the inferior turbinates, treated.

In atrophic rhinitis, there is an increased patency of the nose due to the altered mucous membrane. There is a metaplasia from the normal mucosa to a squamous type of lining with isolated islands of ciliated columnar epithelium. The excessive dryness causes crusting which at times is odorous and is often accompanied by a purulent discharge posteriorly.

The surface of the adenoid is covered with ciliated columnar epithelium which often carries the normal mucus down behind the soft palate in parallel streaks.

After adenoidectomy the nasopharynx is covered with squamous epithelium without cilia. Unless great care is taken in removal of the adenoid, we often find irregular bands of

fibrous tissue which form crypts and pockets where mucus tends to lodge. After ruling out sinusitis, Prof. Hajek, of Vienna, taught his students many years ago to curette the nasopharynx to remove redundant tissue for the relief of postnasal discharge. At the time, it seemed a radical form of treatment, but in view of the above facts this appears to be sound advice in well selected cases.

Another common reason for a troublesome catarrh is due to nasal medication. Vasoconstrictors are the chief offenders, especially when used for a longer period than is necessary. According to a Gallop poll survey during the one-week period ending Feb. 24, 1942, an estimated total of 23,000,000 persons spent approximately $11,500,000 on medicines to get relief from the common cold. This amount was exclusive of medical fees and prescriptions. Of this number (23,000,000), about one in every four persons consulted a physician. Advertising slogans stress the use of nasal drops to clear the head and ventilate the sinuses, but completely disregard the after-effects due to vasodilatation.

The repeated constriction, followed by the compensatory dilatation, in time produces a lack of tone to the nasal vessels. A vicious circle occurs as the patient tends to use the drops more frequently because they lose their effectiveness. The continued use of saline drops causes an excessive dryness of the mucosa, which in itself produces a postnasal discharge. After a time the membrane becomes irritated and swollen, due to the action of the drug. Unless the nasal medication is discontinued, the swelling persists. Accordingly it would appear to be our duty as rhinologists to impress on patients to use sprays or nose drops sparingly and only in acute and subacute infections.

Many eminent rhinologists do not approve of vasoconstrictor drops in acute rhinitis and sinusitis. They point out that the normal reaction to infection in the nose, as in other tissues of the body, is redness and swelling due to the increased blood supply, with an exudation of serum. A diapedesis of cells

occurs and within two or three days the discharge thickens and the infection is localized.

They state that constrictor drops by reducing the engorgement impede the natural defense mechanism. In place of drops, localized heat is recommended in the form of an infrared lamp which aids the normal response and is soothing to the patient.

Unfortunately in the nose the increased swelling due to congestion narrows the airways and produces a fullness and stuffiness which adds to the discomfort of the patient. In order to clear the head, it is necessary to use vasoconstrictor drops which shrink the membrane and give temporary relief. Unless warned by the physician, the patient tends to use drops more frequently and longer than is necessary.

It is doubtful whether nasal medication in acute rhinitis or sinusitis shortens the duration of the infection; however, nose drops when used judiciously tend to open the nose and give the patient considerable relief. In subacute sinusitis, shrinking agents are helpful as they promote drainage and aid in clearing the nose of purulent discharge. When chronic infection is present, attention should be directed to the affected sinus and the use of vasoconstrictor limited to displacement treatment.

Emotional upsets are also responsible for many conditions in the nose and throat. Nasal obstruction causing pharyngeal dryness, with its resultant postnasal discharge, is a common complaint of highly strung young girls, and in women approaching the menopause. When we consider the fact that the ordinary adult swallows on an average of about once a minute, it is easy to appreciate how a person with an unstable nervous system swallows more frequently and develops abnormal feelings in the throat, such as dryness, burning sensations, globus hystericus, etc. Blockage of the nose is a frequent symptom in the later months of pregnancy — the turbinates and membrane resemble the appearance seen in hay fever.

The pale, water-logged tissue is, of course, due to a glandular disturbance and the membrane shrinks with a cessation of the postnasal mucus at the end of pregnancy.

It should be stressed that in the nose the sheet of mucus is one continuous layer which is being constantly replaced, and it moves downward and backward from the nose to the pharynx. Part of it is arrested in the region of the larynx, but the main stream of mucus continues down and coats the inner surface of the esophagus.

In the lungs the sheet of mucus moves upward from the smallest bronchioles to the larger bronchi and then to the trachea. This movement, always toward the glottis, is produced by the action of the cilia. It is common knowledge that secretion in the pharynx is much more irritating to the patient than the same amount of secretion in the nose and as a result nervous or highly strung individuals feel it necessary to constantly clear the throat. In the vast majority of cases the discharge is harmless. When chronic infection is present, the virulence of pathogenic organisms is as a rule attenuated and the acquired immunity developed by the patient tends to render the pathogens innocuous. If infected material is swallowed, the hydrochloric acid of the gastric juice is potent enough to destroy most forms of bacteria; however, with the evidence of inflammation present in the form of increased mucus or tissue edema, it is inevitable that the microbes which are present in varying numbers in all respiratory tracts should be suspected. What evidence do we have of an etiologic aspect? In purulent sinusitis the relationship is undeniable, but in all other conditions this is anything but the case. The mere ability to grow bacteria from the chronic discharges of surfaces open to the air does not in itself prove these bacteria to be responsible. They could just as reasonably merely be taking advantage of conditions created by other means. The wide variety of organisms described in papers on this subject indeed suggests the latter explanation. For example, Hansel in the *Transactions of the American Laryngological, Rhinological and Otological Society, Inc.*, for 1948 quotes Kistner's findings in 400 cases of hyperplastic and nonsuppu-

rative sinusitis. "Positive cultures were obtained in 94.5 per cent of the cases and the streptococcus was the predominating organism."[6]

On the other hand, Grove and Farrior[7] found in sinus washings from 200 similar cases that some type of staphylococcus was by far the most frequent organism encountered. This applied to the sinus mucous membrane as well. In point of fact, it is doubtful whether a bacteriologist would agree to these statements. It would be difficult to convince him that in this type of sinusitis 5.5 per cent are sterile, that the method of collecting antral washing is really satisfactory, or that the finding of staphylococcus in such washings is significant.

During the past few years it has become a recognized fact that in the normal nose nearly 50 per cent of cases contain staphylococcus in smaller or larger numbers, particularly in the anterior half. This is especially true in the presence of obstruction with resultant stagnation of mucus secretion. In the healthy nose the posterior half shows fewer bacteria. The tendency is clearly to find the presence of bacteria in pathologic conditions an attractive possible explanation, whereas the inevitability of bacteria, at least in small numbers, may center attention on them. In many cases of postnasal discharge, it seems highly probable that the bacteria grown from the mucus are saprophytes waiting for the oppprtunity to multiply. Very frequently the germs in these discharges are species of micrococci. These organisms can be found in dust, in the air and in cultures, just as in the nose, and will sometimes give an unpleasant odor; however, these species do not produce frank suppuration, such as boils or abscesses, and they appear to be totally distinct from their relatives, the staphylococci.

Since the cause of the discharge is not due to these organisms, despite their presence in large numbers, their use in vaccines is of doubtful value. This is a common experience and yet unquestionably a great proportion of the population have had such bacterial suspensions given them in desperation; furthermore, if the commonly offered explanation of

bacterial action — that of allergy is correct — then undoubtedly the giving of vaccines may on occasion sensitize patients to bacteria which previously were not guilty of provoking inflammation but which may now do so. In other words, it is not admissible to argue that even if a vaccine does not immunize, that it is safe to give it because at least it does no harm. All these discussions have so far not taken into account the very common virus agents in the upper respiratory tract, particularly those of the common cold and influenza.

There can be no question but that these viruses in a manner not yet understood do permit invasion of the mucous membranes by bacteria, both those normally present on the membranes and those derived from other humans by cross infection. In all probability a very high percentage of so-called bacterial invasions, even pneumonia, are initiated by a preliminary virus.

Also, there is clear evidence that some bacterial polysaccharids may protect the cells of the body against virus infection. The interplay between viruses and bacteria is quite complex and poorly understood at the present time; however, it is easy to understand why it is so important to continue to investigate ways and means of immunizing against these prevalent viruses. Immunity against them is so short after one attack that repeated infection is the rule.

Secondary bacterial invasion is inevitable and as a result is inclined to be moderately frequent. The net result of this over years of time is bound to be a chronic thickening with some dysfunction. The solution to this problem would seem to be in prevention from early life. This prevention may be in virus immunization or chemotherapy of the subsequent bacterial disease.

To sum up, as far as nasal infections are concerned, the object should be to prevent invasion of the mucosa and submucosa with either virus agents or bacteria. Such immunization will never be 100 per cent effective and it will be necessary to supplement immunizing procedures with, first, chemo-

therapy and, finally, with pharmaceuticals designed to offset the action of such microbes which succeed in penetrating and maintaining a foothold in the tissues. In this latter respect, it is significant that the allergic basis for these inflammations is supported by the finding that the antihistaminic drugs will reduce the edema in at least 60 per cent of cases of the common cold in the early stages. This applies particularly in the acute phase when sneezing and watery discharge are the predominating symptoms. Chemotherapy involves an appreciation of the significance of the fact that the mucous membranes under consideration are secreting tissues and that the fluid transfer is, therefore, from the submucosa to the surface, *i.e.*, from the inside outward; therefore, the topical application of antibiotics requires diffusion against the stream of fluid, which mitigates against effective penetration. Under these circumstances, as with lymphatic or glandular infection, systemic therapy is the only route likely to be effective. As far as the virus agents are concerned, immunization is not a practical possibility at present and in any case it appears likely to be short lived. These facts serve to emphasize the need for chemotherapy of virus diseases. The intense research activity in this field has already produced at least two very promising antiviral antibiotics in aureomycin and chloromycetin (aureomycin is formed by a member of the hay bacillus family and chloromycetin by an organism allied to the one which makes streptomycin).

An attempt has been made in this paper to provide a background for understanding the nature of postnasal discharge. It is realized that many other contributing factors have been omitted.

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MISSISSIPPI VALLEY MEDICAL SOCIETY MEETING

AT ST. LOUIS, SEPT. 28, 29, 30.

The Fourteenth Annual Meeting, Mississippi Valley Medical Society, will be held at the Jefferson Hotel, St. Louis, Sept. 28, 29, 30, under the presidency of Dr. Alphonse McMahon, associate professor of medicine, St. Louis University. Over 30 clinical teachers from the leading medical schools will conduct this great postgraduate assembly, whose entire program is planned to appeal to general practitioners. There will be some 60 scientific and technical exhibits, noon round table luncheons, etc. *No registration fee* will be charged and every ethical physician is cordially invited and urged to attend. The entire program and all exhibits will be held on the mezzanine floor of the Jefferson Hotel. The American Medical Writers' Association will hold their annual meeting at the hotel on Sept. 28 and the Missouri Chapter of the American Academy of General Practice on Sept. 30. Programs of all the meetings may be obtained from Harold Swanberg, M.D., secretary, M.V.M.S. and A.M.W.A., 209-224 W.C.U. Building, Quincy, Ill.

THE IMPORTANCE OF SINUSITIS IN ALLERGIC MANIFESTATIONS.*

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The importance of chronic sinusitis as an etiologic agent in the cause of allergic manifestations is still a much disputed question. I am, therefore, very glad to participate in this meeting of your Section, that I may present a few of the facts as we see them, to prove that any intelligent treatment of these manifestations requires an accurate knowledge of the pathology in the paranasal sinuses and of the basic principles of allergy.

After 18 years of close contact with the allergy clinic of the Roosevelt Hospital in New York City, I am convinced that the importance of the disease of the sinuses in the treatment of allergic manifestations is too often under-evaluated by the allergists and also by the otolaryngologists. The patient is thus made to suffer unnecessarily by such neglect in diagnosing and treating sinus disease. Some allergists are prone to say that the sinus disease as found in the allergic patient is the result of their allergic manifestation such as asthma and not the cause of such manifestation. They would throw up their hands and exclaim that these patients have been operated upon time and time again without any cure of the asthma or the sinusitis. I think this is a too pessimistic and unfounded attitude, and I hope to be able to clarify a few of the misconceptions and to prove that excellent results can be produced by careful, intelligent handling of disease of the paranasal sinuses.

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It is necessary in the first place to establish the fact that there is a definite pathology in the sinuses, and in the second place to decide if this pathology is an etiologic factor in the production of the allergic symptoms. I agree that quite often these patients are operated upon without sufficient pathology being present in the sinuses and, therefore, no relief is produced. Many patients are also operated upon in whom this pathology when present is not influencing their allergic symptoms, and likewise the result is bad. For instance, a patient whose asthma is due to pollens, house dust or animal danders, or a patient whose urticaria is due to the ingestion of certain foods or to a contact exposure will not be helped by any operation upon the sinuses. On the other hand, if the patient's asthma or urticaria is precipitated at certain times, or is made worse by upper respiratory infections involving the sinuses, then such an operation will be of value.

Thus, to discuss the topic in hand intelligently, it is necessary to remember these facts, namely, that certain allergic manifestations are of the atopic or skin-sensitive type, while others are of the nonsensitive or infective type. The first group is diagnosed by the skin tests, whether by the intradermal or scratch method, the patch test and the use of elimination diets. The second group is usually negative on skin testing and the presence of pathology in the upper respiratory tract as well as the influence of such infection on the allergic symptoms can be demonstrated. We might add that in addition to the skin-sensitive and the infective types, we may have the combined type, that is, the patient may show positive skin reactions and also the presence and influence of foci of infections. If these three dicta are kept in mind, I think one is in a better position to evaluate the importance of otolaryngological pathology in the treatment of allergic disease.

According to Adam, Herck, of Freiberg, in 1844, first recognized the importance of sinus disease as a cause of asthma; however, it was not until 1872 that an Italian, Voltolini, showed the effect of sinus surgery on asthma when he

reported 11 cases of asthma in which relief followed polypectomy. Since that time, the frequency and importance of sinusitis in asthma has been argued pro and con and its treatment has varied from conservative to radical surgery, although many physicians still advocate no surgery at all.

In order to express our belief in the importance of sinusitis as a cause of asthma and the necessity for the treatment and if possible the elimination of the sinusitis, I shall discuss the incidence of sinus disease in the asthmatic patients, the pathology and the bacteriology of the sinus membranes, the diagnosis of the sinusitis and the operative results.

The literature on the frequency of sinus disease in asthma patients shows a wide variation. This is due in large part to the method of diagnosis, that is, whether it is based on rhinoscopy alone or Roentgenograms. Gottlieb found sinusitis present in only 26 per cent of 117 patients with asthma. Dundas-Grant stated that 63 per cent of 107 cases of asthma had sinusitis. Chobot, in a group of 84 children up to 15 years of age, found X-ray evidence of sinus disease in 67 per cent of the patients. Kern and Schenck, after a careful study of 400 cases of asthma, found that 70 per cent had clinical sinusitis and 87.5 per cent showed X-ray evidence of disease. More recently, Kelley reported that 89 per cent of 100 cases of asthma had rhinologic and X-ray evidence of sinusitis.

In a comprehensive study in our clinic, based on 688 cases of asthma, Cooke found sinus disease to be the cause in 45 per cent of all patients between the ages of 10 and 80 years. Cooke, in this group of 688 cases, found that there was a positive antecedent history of allergy in 45 per cent of the entire infective group and 52 per cent of the atopic ones. Sixty-six per cent of 195 cases of asthma gave an antecedent history of asthma and of other allergic manifestations in 34 per cent. Clarke, Donnally and Coca, however, quoted 80 per cent with a positive antecedent history. Kern and Schenck after a survey of 345 cases of nasal polyposis came to the conclusion that an allergic history or other allergic manifestations are always present.

The pathology of the nasal and sinus membranes in asthmatic patients is well recognized. In our experience almost 100 per cent of the pathology is of the hyperplastic or thickened membrane type as contrasted with the suppurative type. There may be exacerbations of infection and pus appears in the washings and neutrophiles predominate. Polyposis occurred in from 30 to 35 per cent of our cases, but more recent studies have shown this percentage to be lower particularly in patients who had not been operated upon previously. Kelley in his study reported 26 per cent of 89 cases had nasal polyps.

We have also had these membranes cultured, and found that 80 per cent showed positive cultures of one or more organisms. When we compared the organisms obtained from 87 membranes with those grown from previous washings of the antra, we were surprised to find that 43.7 per cent were different from any recovered preoperatively, including the negative ones, and I would like to state here that we do not believe that the simple washing of an antrum and finding it clear and the report of a negative culture of the return flow indicates a normal or noninfective sinus.

The tissues removed from the sinuses were stained and in over 85 per cent bacteria were demonstrated very definitely, while the remaining 15 per cent were too questionable to be considered positive.

Kistner, in 1931, studied 400 sinus membranes culturally and found streptococci present in 94.5 per cent of the cases. Rosenow, of the Mayo Clinic, checked some of his specimens microscopically and agreed that the membranes were infected. Hansel, Piness and Miller, Ashley and Frick, and Balmer and others believed that organisms in the membranes are secondary invaders, but from our own studies we cannot accept this explanation.

The question arises, how is one going to diagnose this type of sinusitis in allergic patients? The answer is, by careful clinical laboratory and X-ray examinations. The history of

frequent colds, stuffiness, nasal discharge anteriorly or posteriorly, sneezing and headache is important, but frequently all of these symptoms are absent. The nasal examination should include anterior rhinoscopy followed by the use of the nasopharyngoscope. The nasopharyngoscope is very important, as otherwise discharge from the antrum, posterior

TABLE 1. RESULTS OF SURGERY ON THE SINUSES IN 200 CASES OF ASTHMA.

Postoperative Years	Cases	Improvement		
		+	++	+++
½-1	18	8	8	2
1-2	23	9	8	6
2-3	18	7	6	5
3-4	24	10	9	5
4-5	17	3	9	5
5-6	11	5	1	5
6-7	7	4	3	2
7-8	8	2	6	8
8-9	17	2	4	3
9-10	22	6	5	1
10-11	9	1	0	4
11-12	16	1	7	2
12-13	15	3	3	2
13-14	8	1	2	3
14-15	6	0	2	4
15-16	6	0	3	3
16-17	2	0	1	1
Total	200	62	77	61
			138 or 69%	

+Indicates slight or no improvement.
++Indicates definite improvement.
+++No asthma or a rare attack.

ethmoids and sphenoids and small polyps posteriorly may be overlooked. Transillumination should be done, but if this reveals nothing abnormal, the examination is not complete. Good Roentgenograms are very important, especially for diagnosing polyps and cysts of the antrum.

In recent years, we have been making more use of radio-opaque solutions in the diagnosis of the type of sinusitis found in our allergic patients. The plain films may often show only slight pathological changes in the sinuses, especially the antrum. These changes often may not seem sufficient to justify an operation. In the infective type of allergic manifesta-

TABLE 2. RESULTS OF SURGERY ON THE SINUSES IN 200 CASES OF ASTHMA BASED ON THE COMPLETENESS OF REMOVAL OF ALL DISEASED TISSUE.

Postoperative Years	Complete Surgery			Incomplete Surgery		
	+	++	+++	+	++	+++
½-1	4	5	1	4	3	0
1-2	6	7	5	2	2	2
2-3	0	6	3	7	0	2
3-4	5	7	3	6	1	2
4-5	3	9	5	0	1	0
5-6	3	0	4	3	0	1
6-7	3	2	1	3	0	1
7-8	0	6	5	2	0	2
8-9	0	4	3	2	0	0
9-10	2	5	1	4	0	0
10-11	1	0	3	0	0	1
11-12	1	6	2	0	1	0
12-13	1	3	1	2	0	0
13-14	1	2	3	0	0	0
14-15	0	2	4	0	0	0
15-16	0	3	3	0	0	0
16-17	0	1	1	0	0	0
Total	30	68	48	35	8	11
		116 or 79.9%			19 or 35.2%	

tions, where it is necessary to eliminate all infection, we have resorted to the injection of radio-opaque solutions such as lipiodol into the sinuses. It seems to us that in these cases the iodized oil, being so opaque, often obscured polyps or cysts if they were in such a position as to cause little defect in the marginal outlines of the sinuses. It was not until 1938 that we discovered a new substance for diagnosing such cases. We

had a patient who was sensitive to iodine, so we decided to try 25 per cent colloidal solution of thorium dioxide. We were impressed with the delicacy, if one may call it such, of the shadows on the X-ray films. The solution, when diluted one or two times, seemed to produce a less dense shadow and the margins and backgrounds of the sinus were more clearly

TABLE 3. RESULTS OF SURGERY ON THE SINUSES IN 200 CASES OF ASTHMA BASED ON THE TYPE OF ASTHMA.

Postoperative Years	Infective Type			Combined Type		
	+	++	+++	+	++	+++
½-1	5	4	0	3	4	2
1-2	8	3	2	1	5	4
2-3	3	5	3	4	1	2
3-4	4	4	2	6	5	3
4-5	3	1	1	0	8	4
5-6	4	1	1	1	0	4
6-7	3	2	1	1	1	1
7-8	2	5	2	0	1	6
8-9	1	3	0	1	1	3
9-10	5	4	0	1	1	1
10-11	1	0	0	0	0	4
11-12	0	4	2	1	3	0
12-13	1	2	1	2	1	1
13-14	1	1	1	0	1	2
14-15	0	2	3	0	0	1
15-16	0	2	2	0	1	1
16-17	0	1	1	0	0	0
Total	41	44	22	21	33	39
		66 or 61%			72 or 77.4%	

delineated. Since that time, we have injected over 500 sinuses without any untoward reactions.

If the X-rays show evidence of the sinusitis, we usually do a lavage of the antrum or sphenoid sinuses. The return flow is centrifuged and examined microscopically for evidence of pus cells and eosinophiles. We usually make cultures also for possible use as vaccines.

Once the diagnosis of sinusitis is made, we must decide upon the proper form of treatment. If polyps are present in the ethmoids or sphenoids, they should be removed if the asthmatic condition justifies such a procedure. By removal of the polyp we do not mean a simple polypectomy but an exenteration of the ethmoids or sphenoids. Polyps, cysts and extensive thickening of the membrane of the antra require a Caldwell-Luc operation. We have not been satisfied with the results obtained by doing simple antrostomies or "window" resections. In conjunction with these operations submucous resections and trimming the turbinates are done for the effect upon the sinus infection and not as a direct benefit to the asthmatic condition. The removal of infected adenoids, tonsils and teeth is also important when indicated.

Regardless of the degree of sinusitis, a complete allergic testing and treatment is necessary. A large number of recurrent polyp cases can be cured if the dust or other inhalant allergens are treated before and after the operation. Blood examinations including Wassermann, kidney and chest examinations, including X-rays and sputum for tuberculosis, molds and spirochetes are usually indicated. Determination of increased sedimentation rate is often important in studying these infective cases. Recently the question of vitamin deficiency has been emphasized, and in certain cases vitamin assay is necessary.

In discussing the effects of sinus surgery upon asthma, I would like to refer first to the frequency of the various sinuses involved and the operations performed. The antrum is the most frequently involved and the ethmoids second. The frontal sinus is the least often involved. The most frequent operation is the exenteration of the ethmoids, with the Caldwell-Luc operation on the antrum being next in order. The external operation on the frontal sinuses is rarely necessary. Usually after performing an exenteration of the ethmoids and a Caldwell-Luc operation, the frontal sinus is provided with better drainage and ventilation through the frontal duct and exacerbations of infection are infrequent.

It seems to us that the importance of sinus disease as a primary cause of asthma is well shown by the effects of sinus surgery in asthmatic patients even after many years' duration. Unfortunately, the results as recorded in the literature are not too encouraging.

Heatley and Crowe in a series of 62 operative cases found 53 improved, but "only one case was enthusiastic enough to describe himself as cured" after three years. Rackeman and Tobey studied 91 cases of asthma whose sinusitis was treated surgically and reported 14 per cent cured, 46 per cent improved, 31 per cent unimproved, and 9 per cent died. Weille stated that in his group of 40 patients, whom he operated upon for sinusitis, only 50 per cent had a chance for relatively long confirmed improvement in their asthma.

Schenck and Kern, Vaughan and Warner, and McGregor reported even less favorable results. It seems to us, however, that in many of these reports it is not made clear as to the importance of associated sensitizations, the extent of the sinus pathology, and the completeness of the removal of the pathology from all of the sinuses.

Since 1935 we have made six very complete studies of the results of our sinus surgery in asthmatic patients. The first study, reported in 1935, included 120 cases, the longest of which had been followed for three and one-half years. In 1947, I reported on 200 patients with a postoperative period of observation of 17 years. It was very interesting that the percentage of improvement in the asthma was almost the same in the six groups. Any intelligent analysis of the effect of sinus surgery on asthma must include accurate information as to the type of asthma, that is, whether due to infection alone, infection combined with sensitization or to sensitizations alone. In the second place, it is necessary to know how many sinuses in the infective type of asthma are involved with disease and how completely was this pathology removed by operation. In other words, if the ethmoids and antrum are involved an operation on the antrum or ethmoids alone will not produce a good result. Likewise, if both antra are

diseased and only one is operated upon there is no reason for thinking the patient will be cured. Complete elimination of pathology as far as possible is absolutely necessary. For this reason we do not believe that the simple removal of a polyp or polyps with the snare or making of a window in an antrum are sufficient operative procedures to cure asthma. I have three slides analyzing our last study of 200 asthmatic patients whose sinus disease was treated surgically. They have been followed postoperatively for one-half to 17 years. They have all been studied as to the type of asthma and the completeness of the surgical procedures in removing the pathology. We are not reporting any results under a six months' follow-up because these results are too transitory; the effect of the anesthetic often produces a temporary relief from the asthma, while the removal of the infected sinus membranes may produce an immediate exacerbation of the asthmatic symptoms.

The infective group comprises 107 cases and the combined group, that is, infestion plus sensitizations, 93 cases. The groups in whom sinus surgery was considered complete had 146 cases and the incomplete group 54 cases.

The improvement in the total group was 69 per cent. This is about the same figures reported in our previous surveys. In this group, however, we are including all of our clinic patients who are more difficult to treat as thoroughly or as completely as private patients.

Several points should be stressed in this analysis. In the first place, it is quite obvious that there is a striking difference in the group who were operated as completely as possible and the group treated incompletely; 79.9 per cent improvement as compared with 35.3 per cent. It is also observable that in the completely operated group the improvement increased with the duration of the postoperative period of time. We believe that this is explained by the fact that there are secondary foci of infection in the cervical and bronchial lymphatic glands and the bronchial mucosa and that these secondary foci heal slowly after the removal of the primary focus. It is possible that the patients who did well immedi-

ately after operation never had these secondary foci; while, on the other hand, the patients who did not do well, even with increasing time, lacked the capacity to heal these foci.

In 1941, I reported in the study of 200 cases of asthma, who were operated upon, that 27 of the patients had a "window" resection or an antrostomy with only 40 per cent improvement in their asthma. On the other hand, 19 patients who had had previous antrostomies were operated upon radically and a 79 per cent improvement was produced. Ninety-four cases who had primary radical antrum operations showed a 75.5 per cent improvement in their asthma. It seems that these figures prove that the antrostomy operation does not eliminate the infection in the membranes completely. We have noticed after the antrostomy that frequently the "window" becomes blocked by polypi, or that it does not function any more, and that upper respiratory infections are more frequent, which necessitates more local treatment of the sinuses and, of course, produce exacerbation of the asthma. The patients who have had the necessary surgery completed frequently go through the upper respiratory infections without any or only a slight flare-up of their asthmatic symptoms.

Referring again to our analysis of the 200 cases, we find that the results are better in the combined type of asthma as compared with the infective type. In this group the infection is often a secondary factor, and, in addition, all of these patients are treated with injections of the indicated allergens as well as by the proper elimination of the allergens when possible. Injections of house dust have not only helped to maintain prolonged freedom from asthma but have in many cases helped to prevent the recurrence of nasal polyps. Practically all of our patients of both groups were treated with autogenous vaccines made from cultures of the infected sinus membranes removed at operation. In addition, all extranasal infections such as molds (fungi), spirochetes and tuberculosis should be treated; infected teeth, tonsils and adenoids should be removed. A few of the patients who improved but moderately after a complete sinus operation showed striking

improvement after having an ovarian or uterine tumor or an infected gall bladder or appendix removed.

I made an additional study of these patients as to the age of onset of their asthma, the duration of the asthma and the age at operation. It seems from these studies that we can say that a patient whose asthma began between 21 and 35 years of age, has been present not longer than 10 years, and preferably less than five years, and the patient is between 31 and 50 years of age at the time of operation has the best chance to obtain a satisfactory result.

SUMMARY.

The incidence of sinus disease in asthmatic patients, the type of pathology, the bacteriology of the diseased membranes, and the results of sinus surgery in asthmatic patients are discussed to show that sinus disease is a very important cause of asthma. Any thorough and intelligent treatment of asthma requires that careful consideration be given to pathologic condition in the paranasal sinuses. Special diagnostic studies such as the use of the nasopharyngoscope and the injection of radio-opaque solutions into the antrum are necessary in all doubtful cases of disease of the sinuses.

In order to obtain accurate information as to the effect of sinus surgery on asthma it is necessary *1.* to study the etiologic factors causing the asthma, that is, whether infective alone or infective associated with demonstrable skin-sensitizations, and *2.* to analyze the type of sinus surgery, as to whether the diseased tissue in the sinuses is removed completely or incompletely.

Immediate improvement should not be expected and follow-up studies have shown that with increasing postoperative periods of time steadily improving results are obtained. We believe that this is due to the gradual elimination of the secondary infections in the cervical and bronchial lymphatic glands and the bronchial mucosa. It is possible that the patients who do poorly lack this capacity to overcome their secondary foci of infection.

Surgical procedures on the sinuses cannot be expected to produce improvement of the asthmatic condition if extranasal infections and associated allergies of the skin-sensitive type are not treated properly. Autogenous vaccines are considered important in the treatment of the infective cases.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary of the Academy, 100 First Avenue Building, Rochester, Minn.

LOCAL RESECTION OF CARCINOMA OF THE RIGHT MAIN BRONCHUS.*

EDWIN N. BROYLES, M.D., and
WILLIAM F. RIENHOFF, JR., M.D. (by invitation),
Baltimore, Md.

The diagnosis and treatment of lung tumors is a brilliant story and tribute to all branches of medicine and surgery. Pneumonectomy, lobectomy and segmental resection require not skilled surgery alone but also expert X-ray, bronchoscopic and medical diagnosis and treatment. Following the publication of the results of surgical treatment of lung tumors both the medical profession and the laity are seeking this expert aid earlier, with definite improvement in prognosis. The early diagnosis of tumors advantageously located (trachea and large bronchi) may warrant the local removal of the tumor, either through the bronchoscope or by surgical resection without sacrificing lung tissue.

The case reported here represents such a combination of favorable development.

Mrs. A. S., No. 466302, white female, aged 22, entered Johns Hopkins Hospital on June 29, 1948, complaining of hemoptysis. Her present illness began in December, 1947, with an insidious onset of fever, vague aching in right chest, cough and expectoration of blood. She was sent to a local hospital, where she was given penicillin. She recovered from the acute condition but had a persistent wheeze, cough, occasional dyspnea and hemoptysis. Six weeks ago she became very dyspneic. She was sent to a nearby city, where she was bronchoscoped on two occasions. Tissue removed was diagnosed carcinoma. Since the bronchoscopic examinations she has been free of cough, wheezing and dyspnea. Because of the diagnosis, her local doctor requested her to consult Dr. Rienhoff.

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She was again bronchoscoped on June 30, 1948, and a small, red, velvety tumor, about the size of a raspberry, was seen in the right main bronchus, 1 cm. from the carina. Tissue was again removed and diagnosed lung carcinoma (S. P. 48-2480). Because of the small size of the tumor and its location, it was thought an attempt should be made to resect the tumor, instead of resecting the right lung and bronchus up to the carina. On July 1, 1948, Dr. Rienhoff, through a right chest incision, exposed the trachea and the right bronchus, after ligating and dividing the azagos vein; this permitted good exposure also of the tracheal bifurcation and the first portion of the left main bronchus. The lymph glands in the region of the bifurcation of the trachea were not involved, and the outer wall of the right bronchus had not been penetrated by the growth. An incision was made in the lower trachea and right bronchus, which exposed the region of the carina and location of the growth. With an elliptical incision, the growth, along with the bronchial wall, was removed and the region burned lightly with an electric cautery. Interrupted silk sutures closed both defects (incision and resection) and the chest wall was closed in the usual manner. Section of the resected growth (S. P. 482496) was diagnosed bronchial adenoma. The postoperative course of the patient required aspiration of fluid from the right chest on several occasions, and there was some fever. Because of the first diagnosis on July 12, 1948, the patient was bronchoscoped and 1200 mc. of radon placed over the site of the resected tumor for one minute. At this time there was no constriction of the bronchus and no evidence of tumor. These have been the findings on later bronchoscopic examinations done in August, October and December, 1948. The patient has remained well. There has been no hemoptysis, no dyspnea and, from examination, no evidence of recurrence of the growth.

Slides of the first tissue removed were obtained; also, slides of our first biopsy were re-examined with the diagnosis still remaining lung carcinoma. It would seem, then, that the epithelium covering the growth had undergone what appeared to be a malignant change, whereas the deeper section of the excised tumor was an adenoma.

With earlier diagnosis, it is hoped that more small tumors of the trachea and larger bronchi may be completely and successfully extirpated.

NATURAL ABSENCE OF PART OF THE BONY WALL OF THE FACIAL CANAL.*

STACY R. GUILD, Ph.D.,
Baltimore, Md.

Injury of the facial nerve is a dreaded complication of otologic surgery. The course of the facial nerve through the temporal bone is, therefore, always emphasized in teaching the anatomy of the region to house officers on otologic services. Precautions to safeguard the nerve during operations are stressed, and young staff members are repeatedly warned that it may be injured even by the pressure of dressings on an accidentally exposed segment.

It seems not to be generally realized, however, that without disease or operation a part of the facial nerve may be devoid of protection by bone. In fact, a survey of textbooks and handbooks of anatomy and of surgery commonly used by students failed to reveal any mention of an absence of part of the osseous wall of the facial canal (Fallopian canal or aqueduct of the older terminologies) not due to some disease process. The books give the impression, rather, that the facial nerve always is covered by bone throughout its course in the temporal bone except when necrosis or erosion of the osseous wall of the canal has been caused by disease.

Experienced otologists, especially those who have examined many sets of histologic sections of human temporal bones, know from observation that the facial nerve in uninfected ears is not always entirely covered by bone, and that a gap in the continuity of the osseous wall may be present in any part of the facial canal. Despite their own awareness of the clinical implications of the observed anatomic condition, they often find it difficult to make assistants fully realize the seri-

*From the Otological Research Laboratory, The Johns Hopkins University School of Medicine.

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ous possibilities. Part of the difficulty arises from the absence of pictorial illustrations in the available literature. The pri-

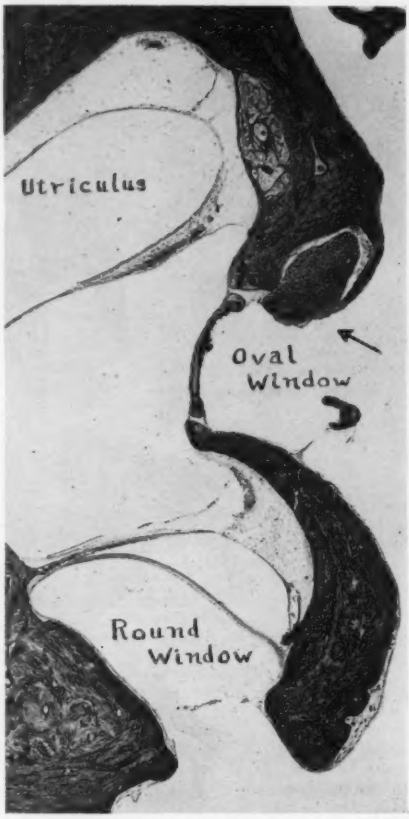

Fig. 1. Photomicrograph, at low magnification, of part of a section of the left temporal bone of a 35-year-old man. The osseous wall of the facial canal above the oval window is incomplete in this ear, so that part of the facial nerve is covered only by fibrous tissue and epithelium. Note that the mucosa of the middle ear is normal. There was no history of ear disease, and the hearing was normal.

mary purpose of the present communication is, therefore, to provide visual evidence that can be used in teaching.

Three examples of gaps in the continuity of the bony wall of the facial canal are shown in the accompanying photomicrographs. One of the gaps shown is in the part of the

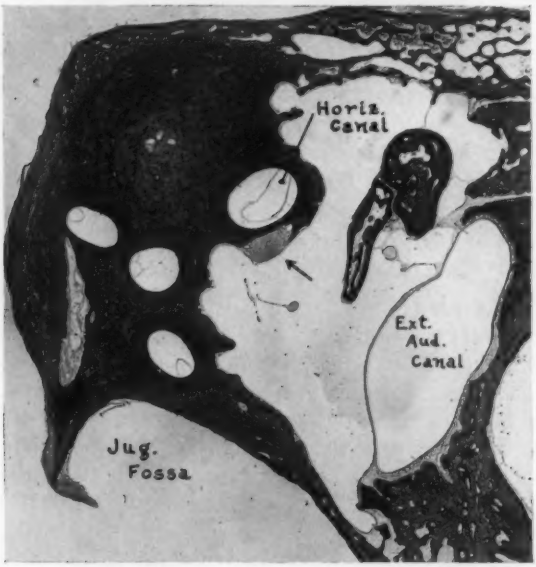

Fig. 2. Photomicrograph, at very low magnification, of a section of the left temporal bone of a 63-year-old woman. Her facial nerve (indicated by the arrow) in the region below the prominence of the horizontal semicircular canal, is covered only by a thin layer of fibrous tissue and by normal middle-ear mucosa. In operating on a patient with a similar gap in the continuity of the osseous wall of the facial canal, especially in doing a fenestration operation via the endaural approach, extreme care in the handling of instruments, suction tips, sponges and dressings would be necessary to avoid injury to the facial nerve.

facial canal that passes above the oval window (see Fig. 1), one is in the part of the canal just below the prominence of the horizontal semicircular canal (see Fig. 2), and one is in the so-called descending, or mastoid, part of the canal (see Figs. 3 and 4). The low magnifications used for the pictures permit, in each instance, easy recognition of the relations of the facial nerve to other structures of surgical importance

but do not portray well the histologic detail of the canal wall in the region of the defect. All of the specimens are from adults, and in no instance does the histologic appearance, at

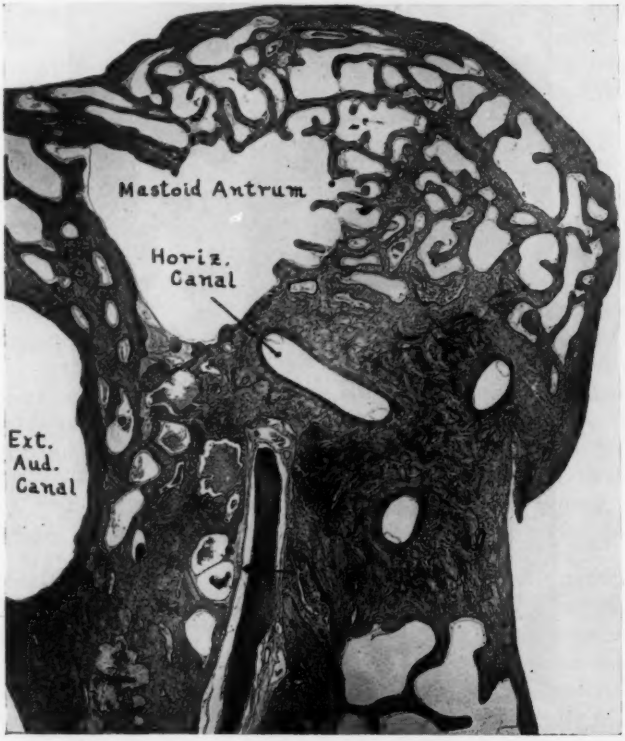

Fig. 3. Photomicrograph, at very low magnification, of a temporal-bone section of a 25-year-old man, showing almost the entire length of the descending, or mastoid, portion of the facial nerve. The arrow drawn across the facial nerve indicates a region which has no bone between mastoid cells and the nerve.

any magnification, give reason to suspect that a bony wall was previously present or that any bone of the region has been destroyed. The gap in the bony wall of the canal is bridged simply by ordinary fibrous connective tissue, which

is covered on the surface toward the middle-ear cavity, or toward the mastoid cells, by the epithelium normal for the region.

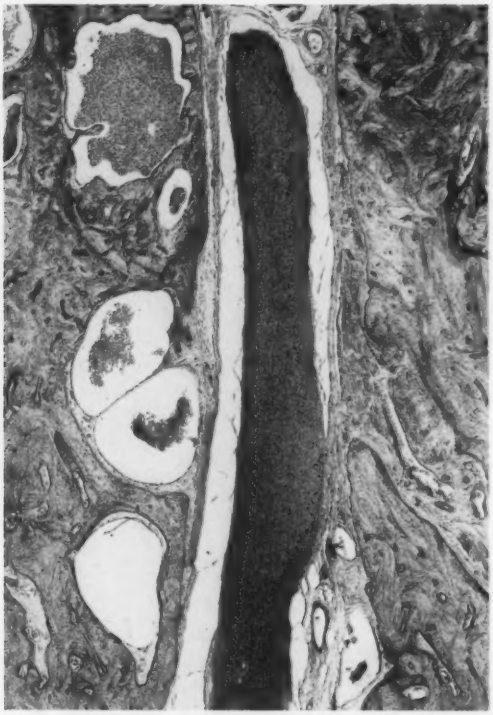

Fig. 4. Photomicrograph of part of same section as is shown in Fig. 3, at a magnification sufficient to show the structure of the wall between some of the mastoid cells of this ear and the facial nerve. The mild infection present was a terminal event and was not the cause of the gap in the continuity of the bony wall of the facial canal.

Large gaps of the bony wall of the canal, *i.e.*, large for the region, have been used for the illustrations, both in order to make the condition easily recognizable in low-magnification photomicrographs and to make obvious the potential dangers in case of an otologic operation on a patient with a similar

anatomic condition. Overgrowth of such a defect of the bony wall by granulation tissue would hide it from direct observation and thus further increase the risk of operative and of postoperative trauma to the facial nerve. It is quite possible that many instances of facial paralysis have resulted from failure to recognize the presence of a gap in the continuity of the bony wall of the facial canal. After a gap in the bony wall is recognized, proper precautions can, of course, be taken to avoid injury to the underlying nerve.

The incidence of gaps in the continuity of the bony wall of the facial canal has not been determined, either for large defects such as are shown in the illustrations or for the more numerous smaller defects. The impression gained by the writer, during the course of other studies of the laboratory's collection of temporal-bone sections, is that the frequency of occurrence is sufficient that the condition should be regarded as a variation rather than as an anomaly. At least a dozen ears of the collection have large osseous gaps, of a size comparable to that shown in Fig. 1, in the canal wall above the oval window; and for that region the writer believes the total incidence of gaps, of one size or another, is as high as 15 per cent. The incidence is probably lowest in the descending, or mastoid, part of the canal.

Bilateral symmetry of defects in the bony wall of the facial canal has been observed in numerous instances, including both the cases shown in Figs. 1 and 2, but a systematic survey of the sectioned material of the laboratory has not been made from this standpoint. If bilateral symmetry of the condition described is the rule, rather than the exception, heredity must be regarded as an important etiologic factor.

In the individual case, it is quite impossible to know in advance of an operation whether or not part of the facial nerve lacks bony protection. Every otologic operation should, therefore, be conducted in such manner as to avoid injury to the facial nerve if an unexpected gap in the continuity of the bony wall of the facial canal is actually present in the patient's ear.

HEARING TESTS AND TESTING.*†

STACY R. GUILD, Ph.D.,
Baltimore, Md.

Hearing tests are numerous and varied. Tests have been made by means of the spoken voice, by whispers, by tuning forks, by watch ticks, by acumeters, by clicking two coins together, by the Galton whistle, by Koenig bars, by monochords, by audiometers and by other means. Tests of ability to hear also include pitch discrimination tests, fatigue tests, loudness-balance tests at a series of intensities, tests made with masking of the opposite ear, tests made with a masking sound introduced into the ear being examined, monaural and binaural tests, so-called open-field tests and tests with headphones, tests for malingering, tests for psychogenic deafness, etc., etc.

In most instances, the brief phrases in the preceding paragraph represent not merely one hearing test but what may be called families of tests.

Tests made with tuning forks, for instance, include the Weber test, the Schwabach test and its modifications, the Rinne test and its modifications, the Gellé test, the Bing test, the Fowler test, the Lewis test, the Frederici test, the Stenger test and others to which names are attached, besides the use of forks over a range of seven octaves or more to determine thresholds of hearing.

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*Presented, by invitation, at a Symposium on Problems of Deafness, held under the joint auspices of the New York Academy of Medicine, Section on Otolaryngology, and the New York League for the Hard of Hearing on Feb. 16, 1949.

†From the Otological Research Laboratory, The Johns Hopkins University School of Medicine.

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Voice tests of hearing, likewise, are manifold. They are made with the so-called live voice and with the recorded voice, and each of these is used for threshold tests and for tests of understanding at intensities above threshold. The material used in voice tests of either type may be entire sentences or it may be numerals, or familiar words chosen for the pitch range of the essential sounds, or lists of nonsense syllables, or lists of phonetically balanced words, or lists of spondee bisyllabic words, etc. All these voice tests may be administered monaurally or binaurally, by headphones or by open-field techniques, and with or without the use of masking.

The phrase, *audiometer test,* is another example of including many test procedures under one heading. Not only does each audiometer test consist of a series of tests at different tonal frequencies but audiometers actually used in various parts of the world differ from one another in basic construction and in the calibration units used (decibels and Nepers and arbitrary values, such as in the Bunch-Dean instrument). The method of charting observations differs; for some audiometers normal hearing is recorded as a straight line, for others as a curved line. Some audiometers generate discrete frequencies at fixed intervals, others generate a continuous range of frequencies. With one of the latter type, recently described, the patient makes his own audiogram by turning a control dial back and forth according to instructions given before the test is started. Audiometers are used for both air-conduction and bone-conduction threshold tests, with and without masking, for loudness balance tests, for fatigue tests, for tolerance tests, and for group testing as well as for individual tests.

The above list of hearing tests, though incomplete, makes it obvious that an adequate presentation of the topic assigned to me in this symposium would require more time than is available. In fact, hours of time would be needed to present comprehensively any one phase of the topic; either to describe each hearing test, or to discuss the merits of each test, or the way each test should be performed, or the possible sources of

errors in each technique, or the possible interpretations of the results of each test, or the evidence that the interpretations customarily made of each test are actually warranted by the facts. It is, therefore, necessary that the presentation of the topic this evening be limited mostly to statements of general principles and conclusions.

The reasons for making hearing tests are many. They range from a desire to determine fitness for a given occupation, such as a school child or a soldier or a telephone operator, to attempts to gather data on which to base, or to modify, the design of complicated electrical apparatus, such as a telephone circuit or a recording instrument. Hearing tests are sometimes made merely to impress or to satisfy patients who have read popular articles about the scientific wonders of the age and who accordingly feel they have not had a good examination unless a lot of gadgets are used. The usual reason an otologist makes hearing tests is to get information that may help determine why the patient has impaired hearing and whether any treatment is indicated; occasionally he wishes also to learn approximately how much impairment of hearing the patient has, for comparison with later tests.

Whatever the reason may be for making hearing tests, the tests to be used should be chosen to yield the desired information. For instance, if one wishes to know how well a patient understands speech, voice tests should be used, not a series of pure tones. The otologist, confronted by the multiplicity of hearing tests, often has difficulty choosing the best ones to use. This difficulty is not new, though we of the present day may sometimes think it is. Over a half-century ago, Bezold, who himself originated a series of test procedures, wrote about the need to limit hearing tests to the purposeful ones and complained about time-consuming unnecessary tests. The advent of oscillating electrical circuits and electronic amplifiers has merely accentuated the dilemma faced by practicing otologists.

Whatever the reason may be for testing the hearing, and whether or not the tests chosen be simple ones or ones that

require the use of complicated apparatus, the tests made, if they are to have any value, must be made carefully and must be made under proper conditions. In spite of all that has been published and spoken about the need for quiet surroundings in which to make hearing tests, and about the importance of masking the better ear when the poorer one is being examined, both of these factors essential to obtaining reliable results are even this year more honored in the breach than in the observance; furthermore, in spite of all warnings, every day of the year defective tuning forks are used, watches of unknown acoustic characteristics are used, audiometers badly out of correct calibration are used, audiograms are made by unskilled personnel, etc. The results of such tests are, at best, misleading. If acoustical physicists and electronics engineers were habitually as careless in making their hearing tests as are many persons who do clinical testing, both in medical circles and in other fields, we would not yet have good telephone circuits, good radios, good phonographs or good "talking movies," except it be by accident.

Mistakes are more apt to be made in using electrical apparatus or complicated techniques than in making simple tests. When the data from the various tests made of a patient's hearing are not consistent with each other, the inclination should, therefore, be to trust the impressions gained from the simpler tests, provided there is no reason to suspect malingering and no opportunity to repeat the tests. The best procedure, of course, is to repeat the tests after ascertaining that the apparatus actually is in proper operating condition in all respects and is being properly used. With cooperative patients such a repetition of the tests usually eliminates the supposed discrepancies and results in confirming the impressions given by the simpler tests of the first examination.

Inconsistencies between parts of a group of tests, or between successive tests on different occasions, are often regarded as indicating either malingering or the presence of a psychogenic factor in the impairment. The inconsistencies may, however, be due to nothing more serious than the fact

that the patient has "auditory after-images." Experienced otologists are familiar with this phenomenon, but oftentimes their assistants, who actually make the hearing tests, are not aware of the possible complicating factor. Accordingly, the test is made at a speed that is all right for patients who do not have "auditory after-images" of faint sounds but that is too fast for patients who do have this annoying condition. When the examiner of such a patient forgets to vary the intervals between interruptions of the test tones, he may be misled into thinking the patient's threshold is normal when it actually is impaired as much as 55 or 60 db. The potential harm to the patient of such an error is considerable, especially if the inconsistencies between tests are interpreted as indicating malingering or psychogenic deafness.

The data obtained by the making of hearing tests, whether they be simple or complex tests, are merely marks on paper until interpreted. The interpretations desired may be in terms of placement in school, of assignment to a job, of design of a hearing aid, or in terms of the cause of an impairment of hearing. For clinical purposes, the results of hearing tests, no matter how detailed and reliable the tests be, have value only when the data are interpreted in the light of the information furnished by a thoughtfully taken history and a good physical examination. For instance, by no battery of hearing tests can a diagnosis be made of the cause of a conductive deafness, which may be due to impacted cerumen, to otosclerosis, or to some other cause; nor can the hearing tests alone distinguish between the impaired hearing normal for an elderly person but abnormal when found in a child, nor between the possible causes of total deafness. For most patients, a few simple tests suffice to provide all the information needed to supplement the history and the physical examination; more tests do not change either the diagnosis or the decision as to what treatment, if any, is indicated.

If enough were known about the subject of the relation of lesions to impaired hearing, it might well be worthwhile in clinical work to make elaborate hearing tests routinely. Most

of the lesions, however, that cause impaired hearing can be studied only by histologic examination after death. By this method important correlations have been made with hearing losses known to have been present during life, but the number of cases that have been thus studied is too small to warrant conclusions as to the effect on hearing of small differences in lesions of the same general type. In other words, as yet only large differences in hearing can be correlated with differences in the lesions responsibile for the impaired function.

The conclusion is inescapable that the knowledge needed to interpret hearing tests intelligently has not increased as rapidly as have testing techniques. Factual information about the physiology of the organ of hearing, which includes parts of the brain, is as yet so incomplete that for many hearing tests hypothetical interpretations only can be made with respects to lesions possibly responsible for an impaired function revealed and measured by the tests.

The gaps in our knowledge are at present so great that a clinician might well apply the following basic questions to each proposed hearing test: *1.* What may I learn, that I do not already know, by making this test? *2.* How good is the evidence that the interpretations made of data obtained by this tests are correct? *3.* If correct, what value do the interpretations made of the data from this test have in the care of patients? In other words, the clinician should make a clear distinction between tests for research purposes and tests for clinical purposes, and he should recognize clearly that nothing is gained clinically by the making of hearing tests that cannot be interpreted with reasonable certainty into terms of causative lesions. All too often a diagnosis, so-called, amounts to nothing but a statement of the kinds of hearing defects found by the tests made. Such a statement, no matter how elaborate the battery of tests used or how detailed the report of the data obtained, is merely a description, and not an interpretation, of hearing tests, and has no medical usefulness whatsoever. The situation constitutes a challenge to investigators, both laboratory and clinical.

A METHOD FOR IMPROVEMENT OF THE CURVED NASAL TIP.

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New York, N. Y.

The reconstruction of the tip of the nose, particularly of the soft parts, is infinitely more complex than those of the hard parts. The difficulties encountered in obtaining a satisfactory end-result in the correction of the nasal tip are the tip, the columella and the nostrils.

At the conclusion of the usual rhinoplastic operation, especially when a parrot-shaped tip is present, the extreme point of the nose may remain too rounded and flat. Although the technical steps of the operation may have been properly executed, as far as the frame work is concerned, there still remains a somewhat curved contour.

To correct this tendency, a small piece of cartilage, more or less rhomboid in shape, 12 mm. in each dimension, is inserted into the subcutaneous tissue of the nasal tip. This cartilage can readily be obtained from the resected alar cartilages or cadaveric cartilage (it is equally as good). If the pieces are not sufficiently thick, the thin material may be laminated and held together with one plain catgut suture.

Before the columella is sutured, the skin at the nasal tip is freed from its underlying structures along the inner and outer horns of the lower alar cartilages. The columella is then sutured to the septum, employing the so-called orthopedic stitch. The small rhomboid of cartilage that has been previously prepared is introduced beneath the skin at the extreme

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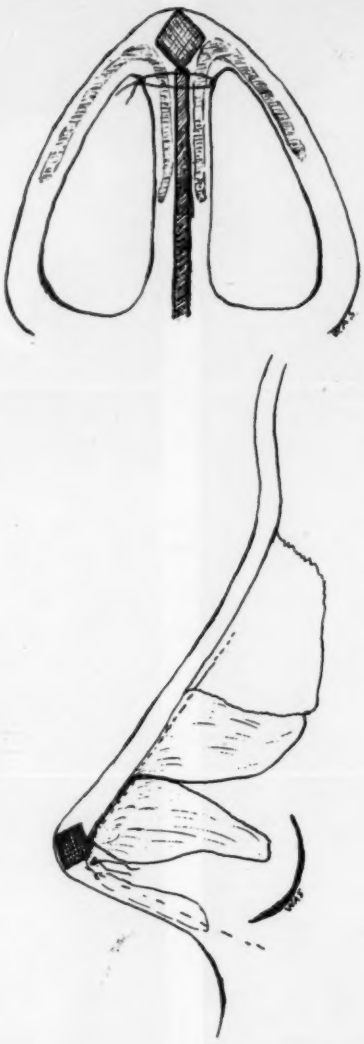
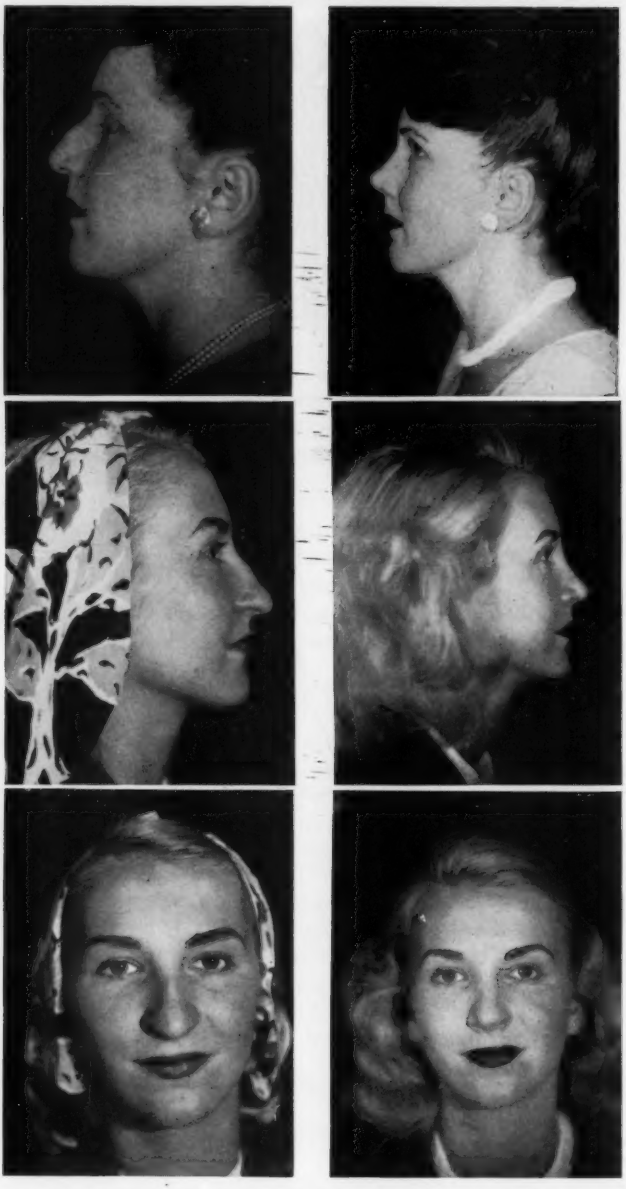


Fig. 1. The cross-hatch area represents the position and relationship of the rhomboid of cartilage.



Figs. 2 and 3 exemplify the results obtained from the application of this technique.

tip of the nose, with its longitudinal axis corresponding to that of the nose. It comes to rest at the uppermost part of the columella. When the rhomboid of cartilage is in place, a mattress suture is passed through the columella at its uppermost part. This suture secures the rhomboid in its new position. If there is any doubt that the morsel of cartilage is not secure, an alternative method of fixation may be employed. A fine nonabsorbable suture may be passed through the rhomboid. With a needle affixed to each end, the suture ends are passed through the skin of the nasal tip in such a way as to secure the rhomboid in the desired position.

The tip assumes a pleasing retrousse effect instead of the ordinary flat tip. This effect obtained is assured by immobilization of the rhomboid by a narrow strip of adhesive tape placed below the extreme point of the cartilage and another piece above the rhomboid.

As a result of employing the morsel of cartilage (rhomboid in shape), three things are accomplished:

1. The tip of the nose is more definitely defined.
2. The columella is lengthened.
3. The borders of the nares assume a more graceful curve.

JOINT MEETING SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY AND NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

The joint meeting of the South Carolina Society of Ophthalmology and Otolaryngology and the North Carolina Eye, Ear, Nose and Throat Society will be held at Hotel Poinsett, Greenville, S. C., Sept. 12 through Sept. 15. The following program will be presented:

Dr. A. D. Ruedemann, professor of ophthalmology, Wayne University, Detroit, Mich.:

1. The Differential Diagnosis Between Unilateral and Bilateral Ocular Protrusion.
2. The End-Results with New Radium-D Applicator in Comparison to Radon.
3. Ocular Manifestations of Allergy.

Dr. Peter C. Kronfeld, department of ophthalmology, Illinois Eye and Ear Infirmary, University of Illinois, Chicago, Ill.:

1. Differential Diagnosis of Acute Glaucoma.
2. Prognosis of Retinal Detachment.
3. Newer Drugs in Ophthalmology.

Dr. Rudolph Aebli, professor of ophthalmology, New York University, Bellevue Medical Center, Post-Graduate Medical School, New York, N. Y.:

1. The Relationship of Muscle Imbalances to the Palpebral Aperture and Pseudo-Ptosis.
2. Principles of Ocular Muscle Surgery.
3. Congenital Muscle Anomalies.

Dr. Carl C. Johnson, associate, department of ophthalmology, Harvard University, Boston, Mass.:

1. Surgical Treatment of Ptosis.

2-3. The Diagnosis and Surgical Treatment of Glaucoma.

Dr. Preston C. Iverson, New York, N. Y.:

1. Neoplasms of the Lip, Nose and Eyelid and Plastic Repair of the Lesions.

2. Congenital Deformities of the Face.

Dr. Louis H. Clerf, Philadelphia, Pa.:

1. Cough Viewed from Otolaryngologic Standpoint.

2. Paralysis of Larynx: Surgical Treatment.

3. Malignant Neoplasms of Larynx.

Dr. Kenneth M. Day, Pittsburgh, Pa.:

1. Clinical Management of Deafness.

2. Ménière's Disease.

Dr. Russell A. Sage, Indianapolis, Ind.:

1. Diseases of the Mouth and Tongue.

South Carolina Society of Ophthalmology and Otolaryngology: President, Dr. Pierre G. Jenkins; Vice-President, Dr. Murdock Walker; Secretary-Treasurer, Dr. Roderick Macdonald.

North Carolina Eye, Ear, Nose and Throat Society: President, Dr. James Harrill; Vice-President, Dr. G. M. Billings; Secretary-Treasurer, Dr. MacLean B. Leath.

For further information, address Dr. Roderick Macdonald, Secretary, South Carolina Society of Ophthalmology and Otolaryngology, Rock Hill, S. C., or Dr. MacLean B. Leath, Secretary, North Carolina Eye, Ear, Nose and Throat Society.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1949.

Acousticon Model A-100.
Manufacturer: Dictograph Products Corp., 580 Fifth Ave., New York 19, N. Y.

Aurex (Semi-Portable); Aurex Model C-B, Model C-A, Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver).
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model A-1; Otarion Model A-3; Otarion Models A-4 J and S; Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Precision Table Hearing Aid.

Manufacturer: Precision Electronics Co., 850 W. Oakdale, Chicago 14, Ill.

Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone; Radioear Permo-Magnetic Multipower—report not yet published.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Ravox (Semi-Portable).

Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C.

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Orthotronic Model; Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Ralph A. Fenton, 906 Medical Arts Bldg., Portland, Ore.
Secretary: Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Conrad Behrens, 35 E. 70th St., New York, N. Y.
President-Elect: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9-14, 1949.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Rea E. Ashley, 384 Post St., San Francisco, Calif.

Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22 Pa.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Prof. Justo Alonso.

Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, Pa.

Second Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology: Montevideo, January, 1950

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.

Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

Meeting: Mark Hopkins Hotel, San Francisco, Calif., May, 1950.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Secretary-Treasurer: Dr. Victor Goodhill.

Chairman of Section on Ophthalmology: Dr. George Landegger.

Secretary of Section on Ophthalmology: Dr. Harold B. Alexander.

Chairman of Section on Otolaryngology: Dr. Alden H. Miller.

Secretary of Section on Otolaryngology: Dr. Leland R. House.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.

Time: 6 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Alfred Schattner, 115 E. 61st Street, New York 21, N. Y.

Secretary: Dr. Norman N. Smith, 291 Whitney Avenue, New Haven 11, Conn.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

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Vice-Presidente: Dr. Luis E. Olsen.
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Tesorero: Dr. Juan Manuel Pradales.
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